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VOL. 1 NOVEMBER, 1920 No. 3

REPORT ON TETANUS*

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PHILADELPHIA

I. PATHOGENESIS OF TETANUS

The following propositions may be accepted as proved:[1]

A. *The Nature of the Disease.*—The disease is a pure toxemia; the bacilli or their spores may exist indefinitely in the tissues, and no symptoms will be produced unless toxins are formed.

It is important to realize, as pointed out by Tulloch,[2] that at least three different strains of toxic *B. tetani* are recognized, and more than one nontoxic strain. And one must not forget the necessity for symbiosis of the spores of tetanus bacilli (and even of the bacilli themselves) with certain other organisms before a toxin can be produced. Tulloch[3] found *B. welchii* and *Vibrion septique* very valuable in experimental work; while the simultaneous presence in the wound of the pyogenic organisms has long been appreciated as a predisposing factor of the greatest importance.

B. *Tetanus Ascendens.*—In experimental tetanus, when small animals are used, the form known as tetanus ascendens occurs: here the symptoms of the disease begin in the inoculated extremity and, though other neighboring parts may become affected subsequently, yet death or recovery usually occurs before trismus and retraction of the head develop. In the larger animals and in man, however, the symptoms usually begin first in the muscles of the neck and jaws, no matter where the point of inoculation; subsequently the muscles of the back and trunk are affected, and finally the extremities. This form of the disease is known as tetanus descendens.

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*Presented before the Société Internationale de Chirurgie, Paris, July, 1920.

1. The evidence on which they are based is detailed at length in a monograph by Ashhurst and John (The Rational Treatment of Tetanus, Am. J. M. Sc. **145**:806, 1913; ibid. **146**:77, 1913). All references prior to that date may be found there.

2. Tulloch, W. J.: Proc. Roy. Soc., London **40**:529 (Series B) 1917-1919.

3. Tulloch, W. J.: Brit. M. J. **1**:164 (June) 1918.

The explanation of this difference resides in the different methods of inoculation. In experimental animals, the toxin is usually injected into muscle, comes into immediate relation with the motor nerves, is absorbed rapidly by them, and the part of the spinal cord first to be affected is that in anatomic relation with the wounded limb. In man, however, the usual point of inoculation (in civil life, at least) is the hand or foot, and much of the toxin enters the general circulation and is carried to the end-plates of all nerves throughout the body; and the toxin which is thus carried to such short nerves as those of the facial, cervical, and spinal muscles will reach the spinal cord through these short nerves, and will begin to produce tetanic spasm in the parts supplied by these short nerves, even before the toxin in the wounded extremity has had time to ascend the nerves of that extremity as far as the anatomically related region of the spinal cord. Tetanus following wounds of muscular parts in man is not infrequently inaugurated by symptoms of local tetanus.

C. *Course of the Toxin.*—It is a fact that the toxin ascends the peripheral nerves to the spinal cord.

The peripheral nerves have little power of neutralizing the toxin—no more power than has the liver, for instance. After death, toxin may be recovered from the nerves as readily as from the internal organs. The peripheral portions of the nerves are found to give up more toxin than those portions nearer the spinal cord: in other words, the nearer the toxin gets to the spinal cord, the more impregnably entrenched does it become in the nerve tissue; and the readier the access of the toxin to the central nervous system, the less is the amount required to kill.

The toxin ascends the nerves: (1) by way of the axis cylinders (perhaps by means of a centripetal protoplasmic current), and (2) by way of the lymphatic channels of the epineurium and perineurium.

What proportion of toxin ascends by each of these channels does not appear to have been determined. The researches of Robertson[4] and of Teale and Embleton,[5] however, indicate that the lymph channels are of more importance than they were formerly thought to be. Robertson's experiments further indicate that the toxin gains access to the cord through the motor roots.

When the toxin has reached the spinal cord it spreads to neighboring parts of the cord, both to the sensory side and to the neighboring motor regions.

Tetanus dolorosus may be produced experimentally by injecting toxin into the sensory nerve roots; and the animals will die of exhaustion before motor phenomena develop.

D. *Production of Tetanic Symptoms.*—The toxin also enters the general circulation, but only when this toxin reaches the spinal cord does it produce characteristic tetanic symptoms.

In animals, rapid emaciation sometimes follows the administration of tetanus toxin, when no tetanic symptoms are produced; probably these cases, described as *tetanus sine tetano,* are due to the action of the toxin on structures other than the nervous system.

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4. Robertson, H. E.: Am. J. M. Sc. **152**:31 (July) 1916.
5. Teale, F. H., and Embleton, D.: J. Path. & Bacteriol. **23**:50 (Oct.) 1919.

It is probable that most of the toxin which reaches the central nervous system from the general circulation does so only indirectly through absorption by peripheral nerves. Albert[6] contends that most of it is carried directly to the central nervous system by the blood; but he presents no evidence to disprove the former theory, which has recently been confirmed by the researches of Teale and Embleton.[5]

E. *Cause of the Symptoms of Tetanus.*—The toxin stimulates the motor cells of the spinal cord, with the result that the muscles controlled by these cells are thrown into tonic spasm; the toxin also renders the sensory side of the cord extremely susceptible to external stimulus, so that very insignificant stimuli, such as the slamming of a door, jarring the patient's bed, a sudden draught of air, etc., will bring on a clonic convulsion, or at least will greatly intensify, for the moment, the tonic spasms.

II. PROPHYLAXIS OF TETANUS

A. Certain classes of wounds, received in certain surroundings, are more often followed by the development of tetanus than are ordinary wounds. *B. tetani* normally infests the intestinal tract of horses and cattle and is deposited with their dung; it is found in the intestinal tract of perhaps 5 per cent. of mankind. Therefore wounds contaminated with barnyard or highly cultivated garden soil, those produced by dragging in street dust, etc., are especially liable to be infected with tetanus bacilli. Gunshot wounds are liable to contamination not *per se,* but only as these conditions, and others presently to be mentioned, obtain.

Growth of the organisms is favored by anaerobic conditions of the wound. These are present in any wound in which there is tissue destruction, which implies cessation of circulation in the devitalized tissues; the best culture medium for tetanus bacilli is that which contains some dead organic tissue. Contused, lacerated and gunshot wounds offer ideal conditions for the development of any bacilli present; and as the bacilli are carried into the wound only by a missile (shell fragment, fragment of clothing, splinter of wood, rock, etc.) and usually remain attached to this missile, it follows that wounds with retained missiles offer the most favorable conditions possible for the development of tetanus. Punctured wounds are to be dreaded not because the vulnerating instrument is retained, which it seldom is, but (1) because it was contaminated either even before it pierced the skin, or (2) because it carried infection from the skin or clothing deeper into the tissues. Even a superficial brush-burn may give rise to tetanus (as in a case in my own experience) provided the skin or the vulnerating surface carries tetanus bacilli.

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6. Albert: Compt. rend. Soc. de biol. **81**:1127, 1918.

B. Care of the wound is the first step in the prophylaxis of tetanus. It is my own firm belief that efficient care of the wound as soon as possible after its receipt is by all means the most important feature in prophylaxis.[7]

This includes: (1) mechanical cleansing (débridement, extraction of foreign substances, excision of devitalized tissue), and (2) chemical disinfection (3 per cent. alcoholic solution of iodin remains, in my own opinion, the best agent for this purpose; certainly no cauterizing agent should be used, as this will produce more dead tissue).

C. Prophylactic use of antitoxin holds second place, but second only to care of the wound.

The fact that antitoxin is of use as a prophylactic injection scarcely needs discussion. Accumulated experience during the recent war merely served to emphasize its value as already ascertained in civil life. In the War of the Rebellion there occurred, according to Sanford,[8] 505 cases of tetanus among 246,172 wounded, or a little over two per thousand. In the American Expeditionary Forces, during the recent war, according to figures furnished to me by the Office of the Surgeon-General, U. S. Army, there developed thirty-six cases of tetanus among 223,067 wounded, or less than two per 10,000. Even allowing for the vast improvement in the mechanical and chemical disinfection of the wounds, such a remarkable diminution in the incidence of tetanus could never have been secured without antitoxin, especially in view of the highly contaminated nature of the soil in which much of the fighting was done.

Reference may further be made to the communications of Bazy[9] who observed 200 wounded from the same sector, of whom 100 received serum when wounded and only one developed tetanus (the day after the injection), while the remaining 100 received no serum, with the occurrence of eighteen cases of tetanus; of Columbino[10] who narrates that among 200 wounded received in his service, all but two had received a prophylactic injection of serum, and only these two developed tetanus; of Walther[11] whose service received 270 wounded German prisoners, to as many of whom as possible antitoxin was given, with the result that of the whole number nineteen developed tetanus, only one of these cases being in a patient who had received the serum (and in his case the disease appeared the day after the injection); of Fredet[12] who observed twelve wounded German prisoners (to whom the German surgeon left in charge refused to administer proffered French antitoxin, because he believed it to be of no value) six of whom developed tetanus, while among a larger number of serum-treated French (wounded in the same battle, and in

7. The report by Clark (Boston M. & S. J. **176**:541, 1917) of 100 consecutive cases of punctured wounds of the foot, produced by nails, without a single bad result, and the fact that no tetanus antitoxin was employed, indicates the efficiency of prompt and proper care of the wound.

8. Sanford: Internat. A. M. Museums Bull., 1918, No. 7, p. 365.

9. Bazy: Bull. Soc. chir., Paris **42**:2919, 1916.

10. Columbino: Bull. Soc. chir., Paris **42**:2913, 1916.

11. Walther: Bull. Soc. chir., Paris **43**:71, 1917.

12. Fredet: Bull. Soc. chir., Paris **43**:1338, 1917.

an adjoining ward) not one developed tetanus. And especially would I call attention to that graphic representation[13] of the prophylactic value of antitoxin which gives a curve constructed from Sir David Bruce's reports[14] of the incidence of tetanus in the British army, indicating an incidence of from fifteen to thirty-two cases of tetanus per thousand wounded until tetanus antitoxin was administered as a matter of routine to all the wounded, whereupon the incidence fell to two or three per thousand and never subsequently exceeded these figures. Nor against these facts can it be argued that tetanus was prevalent only at the first Battle of the Marne; for as Leriche[15] points out, it was prevalent also in other sectors wherever serum was not available for prophylaxis, and it complicated even clean perforating bullet wounds which neither then nor at any other period of the war were treated by operative disinfection; yet in these wounds as well as in wounds which received operative treatment the incidence of tetanus became negligible whenever serum was available. The fact that the wounded of the British army when cut off in Kut-el-Amara began to develop tetanus, but that the incidence fell at once to its previous low figure when an aviator arrived with a supply of antitoxin, and its prophylactic use was resumed[15]—this, I say, is but another proof of the prophylactic value of antitoxin.

There are three factors to be considered in connection with the prophylactic use of the antitoxin: (1) the quantity to be administered; (2) the site of the injection, and (3) the frequency with which it should be administered.

1. *The Quantity of the Injection.*—The usual prophylactic dose is 1,500 units, U. S. A.

The United States Government's standard method for testing the strength of tetanus antitoxin is: "The immunity unit for measuring the strength of tetanus antitoxin shall be ten times the least quantity of antitetanic serum necessary to save the life of a 350-gram guinea-pig for ninety-six hours against the official test dose of the standard toxin furnished by the Hygienic Laboratory of the Public Health Service." The official test dose of toxin is one hundred times the smallest quantity of toxin which will kill a guinea-pig within ninety-six hours.

Taking the average weight of persons injected as 150 pounds (70 kilograms), and calculating the potency of the antitoxin from the known value of a unit (U. S. A.), it is evident that 1,500 units is a little more than 2,000 times the minimal lethal dose of toxin in such an individual. Such an amount is by no means excessive, in view of the fact that the amount of antitoxin required to prevent death increases in geometric progression with the lapse of time. After the lapse of one hour, twenty-four times as much antitoxin is required as when antitoxin and toxin are injected simultaneously (Remertz, 1911).

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13. Editorial, Arch. méd. Belges **70**:834 (Sept.) 1917.
14. Bruce, David: Brit. M. J. **1**:118 (June 27) 1917.
15. Leriche: Bull. Soc. chir., Paris **43**:76, 1917.

2. *The Site of the Injection.*—Usually it is administered subcutaneously. It is better, however, to administer it intramuscularly, in the immediate vicinity of the wound, in order to flood these tissues with antitoxin even before the absorption of toxin has begun. The antitoxin so injected finds readier access, it is believed, to the nerves of the wounded part, and is admitted to the circulation no less rapidly than when administered by the subcutaneous route.

3. *The Frequency of the Injection.*—The first injection should be given as soon as possible after the receipt of the wound. In military surgery, the first injection almost always can be given some hours before proper care of the wound can be instituted. This fact is fortunate for the wounded man because of the frequency with which operation must be delayed; but in neither military nor civil life does this in any way impair the doctrine that proper care of the wound is the more important of the two factors in the prevention of tetanus.

It is a well demonstrated fact that the antitoxin is all eliminated from the system in about eight or ten days after the injection. Its quantity remains practically undiminished for a week, and then rapidly diminishes to nothing in the course of two days. As the tetanus bacilli, if any are lodged in the wound, may not begin to produce any toxin until at least as long a time as this has elapsed, it is manifestly important that some antitoxin should be present in the system as long as the probability of the presence of toxin persists. In many cases in civil life (vaccination wounds, for instance), tetanus bacilli do not enter the wound at the time of its receipt, but are introduced secondarily by the carelessness of the patient in neglecting to return for dressings and in attempting to care for the wound in septic surroundings at home. But even if present in the wound from the first, the bacilli may lie dormant for weeks in the form of spores, before development occurs with the production of toxin.

The necessity for a second injection of antitoxin about the eighth or tenth day is thus very evident, and I have come to agree with Solieri (1910) who insisted that this injection must be renewed every eight or ten days thereafter until all necrotic tissues are removed and there is a clean granulating surface. The dose should be the same in the later injections as in the first.[16]

16. Vallée and Bazy (Compt. rend. Acad. d. sc. **164**:1019, 1917) have pointed out that the organism utilizes subsequent doses less and less, the greater the time that has elapsed since the first dose; perhaps because it in time develops an anti-antitoxin. So that even if the supply of toxin gradually becomes less as the time elapsed since injury increases, the dose of antitoxin must be maintained at the original amount, or even be increased. The researches of these writers indicate that it may become possible to produce active immunity against tetanus by vaccination with iodized toxin; and that this active immunity may be of more value in cases of late or chronic tetanus than the progressively diminishing strength of passive immunity which is conferred by the serum.

That tetanus may develop after the prophylactic use of antitoxin cannot be denied; but such cases rarely develop very soon after the injury, and when they do appear seem to be less severe than are most cases in which no serum has been administered.

Speed,[17] working in a British base hospital, observed six cases among 12,000 wounded; three of the patients recovered.

Mathieu[18] noted that tetanus which developed after the prophylactic use of serum gave a mortality of 26.6 per cent., while the mortality was 58.1 per cent. among those who had not received antitoxin; while Gessner and Adiger[19] report that among eleven patients who developed tetanus in spite of the prophylactic use of antitoxin (only one injection having been given in each case), there were only four deaths, a mortality of 36.3 per cent.; which is to be compared with the general mortality in the same hospital, which has varied from 64 to 78 per cent. since the introduction of serum treatment. These figures indicate that even a single prophylactic injection tends to reduce the mortality.

These early postserum cases, a class which our French colleagues have happily named "tetanos post-sérique precoce," are certainly rarer than the late postserum cases ("tetanos post-sérique tardif"), and it is undoubtedly due to the nearly universal employment of serum prophylactically that we must attribute the relative frequency during the recent war, of forms of tetanus rarely encountered in civil life: I mean the late, the local, the recurrent and the chronic forms of the disease.

A distinction should be made between late tetanus (that in which no primary attack occurs under four weeks after injury); local tetanus (which corresponds to the experimental form known as tetanus ascendens, except that it never becomes general); chronic tetanus (that which is of long duration, irrespective of other factors; which usually is relatively mild; and which sometimes leaves contractures as a legacy); and recurrent tetanus (in which a primary attack is followed by others). The incubation period scarcely ever exceeds four weeks; and in late or recurrent cases it should be dated from the arousing injury or operation, not from the original wound.[20] Speed[21] has recorded a remarkable instance of recurrent tetanus: A soldier wounded in August, 1916, whose wound healed without operation, received three doses of antitoxin in the first ten days after injury. Chronic tetanus developed in October, 1916; recovery was complete by February, 1917. Recurrence was noted in January, 1918, there having been no new wound and no operation since the original injury. A roentgenogram at this time showed a retained foreign body.

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17. Speed: Med. & Surg., St. Louis **1**:14, 1917.
18. Mathieu: Arch. de méd et pharm. navales **106**:467, 1918.
19. Gessner, H. B., and Adiger, D.: New Orleans M. & S. J. **69**:91 (Aug.) 1916.
20. Hesse: Deutsch. Arch. f. klin. Med. **24**:284, 1917.
21. Speed: Med. & Surg., St. Louis **2**:499 1918.

This leads to a discussion of the prophylactic use of serum before late operations on parts which have been wounded in war or in other circumstances in which contamination with tetanus bacilli is feared. This subject was very fully discussed in 1917 by the Society of Surgery of Paris. As Fredet[22] points out, it involves answers to three questions:

(*a*) What is the frequency and gravity of cases of tetanus occurring late, in spite of early prophylactic use of serum at the time of injury?

(*b*) What is the frequency, nature and gravity of accidents due to reinjections?

(*c*) What is the efficacy of reinjections in preventing late tetanus?

(*a*) To the first of these questions, surgeons have replied by submitting long lists of operations on such wounds with no, or at most with very few, occurrences of tetanus; and the cases that have occurred usually were mild. Fredet himself had only one patient develop tetanus (a mild case with recovery) among about 1,000 who underwent late operation, none of these patients having received a reinjection at the time of operation. But we cannot ignore the significance of certain facts to the contrary, which will be cited subsequently in the discussion of Fredet's third question.

(*b*) It is an accepted fact that accidents ("serum sickness," anaphylaxis) due to reinjections are neither frequent nor serious. Cases of death from anaphylaxis are almost, if not quite, unknown. The fatal case related by Salvatore[23] as one due to anaphylaxis may as readily be attributed to sepsis (local tetanus in case of undrained wound of the elbow joint with retained missile). Accidents such as erythema, urticaria, and even articular effusions undoubtedly are regrettable, and are an annoyance to the patient; but they just as certainly are less annoying than would be the outbreak of tetanus. Moreover, according to certain observations, it is probable that the antitoxin, if administered at the time of operation while the patient is under a general anesthetic, will produce no anaphylactic shock. Acting on the suggestion of Major James F. Corbett, M. C., U. S. Army (who had found it impossible to produce anaphylaxis in experimental animals which under other circumstances were very susceptible to repeated doses of antitoxin), I inaugurated this practice while chief of the surgical service at the Walter Reed General Hospital, Washington, D. C., and a prophylactic injection of antitoxin was administered subcutaneously to each patient while he was under the influence of

22. Fredet: Bull. Soc. chir., Paris **43**:1108, 1917.
23. Salvatore: Gazz. d. osp. **38**:5 (Jan. 4) 1917.

the general anesthetic at the time of any operation on parts which had been wounded in a manner to suggest the possibility of their contamination with tetanus bacilli. No anaphylactic shock was observed in any case. I learned later that precisely the same conclusion as was reached by Major Corbett had been previously reported by Dean,[24] who even with intravenous injections administered to fourteen anesthetized patients (who had already received serum) saw no serious results—one death occurred during the operation, but there was no proof it was due to anaphylaxis; a second patient on coming out of the anesthetic had slight shock, urticaria, and edema of the eyelids; four others had urticaria; and a seventh had edema of the eyelids alone.

(*c*) To the question as to the efficacy of reinjections in preventing late tetanus, very significant, if not conclusive, facts may be submitted in answer. I may mention particularly the experience of Lieut.-Col. John F. Connors, M. C., U. S. Army, who during the war was chief of the surgical service in the Hospital Center at Savenay, France (while I was Group Consultant in Surgery). Colonel Connors writes me that among 48,000 patients who passed through this hospital center only two developed tetanus. The rule was to administer an injection of antitetanic serum to every patient who was to have an operation on parts which had been wounded. The first patient who developed tetanus had received serum at the time of injury, but six weeks later, when Dr. Connors removed a large foreign body from his lung, by some oversight he did not receive another injection of antitetanic serum; he developed symptoms of tetanus within twelve hours following operation, and in spite of active treatment, death occurred in thirty-six hours. In the second case, the patient, with a compound comminuted fracture of both bones of the right forearm from a gunshot wound, had likewise received antitoxin at the time of injury; but as the operation performed at the base consisted merely in manipulation of the fragments and application of a splint, no reinjection of antitetanic serum was given; twenty-four hours later symptoms of tetanus developed, but fortunately after a week of heroic treatment recovery ensued. Col. James P. Hutchinson, who was in command of the A. R. C. Military Hospital No. 1, at Neuilly-sur-Seine, also told me that though it was the rule to administer a reinjection to every patient at the time of a late operation on parts which had been wounded, yet it was found, in the case of two patients who developed tetanus, that this injection, by some oversight had not been given.

24. Dean, H. R.: Lancet **1**:673 (May 5) 1917.

In this connection it is interesting to note these facts observed by Owen:[25] The only case of tetanus which developed among hundreds of operations for old war wounds was in the case of a soldier with nonunion of the tibia, who had had four operations for osteomyelitis, at each of which it was presumed that antitetanic serum had been given, as is the rule in the United States Army hospitals. The last wound was healed by Sept. 1, 1919, and an operation for nonunion (sliding bone inlay) was performed March 1, 1920. No antitetanic serum was given, tetanus developed March 7, and death occurred March 9.

So that, until its uselessness is proved, I believe it is incumbent on surgeons to administer a reinjection of serum to such patients at the time of late operations on parts which have been wounded, and especially if there is a retained foreign body or a dense cicatrix rendering all the more likely the continued but latent existence of tetanus bacilli or their spores.

TREATMENT OF TETANUS[26]

The indications are: (1) to remove the source which supplies the toxin; (2) to neutralize the toxin already formed; (3) to depress the functions of the spinal cord, and (4) to nurse the patient.

A. *Removal of the Source of Supply of the Toxin.*—If this source is known it should be attacked directly: the wound should be widely opened and mechanically cleansed of foreign bodies, sloughs, etc. Then it should be treated with antiseptics, and I believe a 3 per cent. alcoholic solution of iodin is the best. The wound should then be filled loosely with gauze soaked in the iodin solution. I believe the use of any caustic should be avoided, as it favors the growth of tetanus bacilli by the formation of sloughs. If the nature of the case demands it for other reasons, amputation should be performed; then the stump should be left open and treated as the original wound. Probably in many cases it will be well to follow the suggestion of Porter and Richardson to excise the related lymph nodes, particularly if they are palpably enlarged.

In the case of a firmly healed wound, on the other hand, it probably will prove more detrimental to the patient to undertake any formal operation than to leave *in situ* deeply placed and apparently well encapsulated foreign bodies.

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25. Owen: Mil. Surgeon **47**:42, 1920.

26. The importance of recognizing the disease promptly can never be overemphasized. Premonitory symptoms must be recognized, and heroic treatment instituted without an hour's delay.

B. *Neutralization of the Toxin.*—To neutralize the toxin the best remedy is antitoxin.

As it may possibly still be thought by some that antitoxin is of no value in treatment, I can only recommend to these doubters to do what Delbet, himself formerly a doubter, advised, namely to study the subject. To any one with an open mind, I believe the evidence, though circumstantial and not perhaps positive, will nevertheless seem convincing. Among many other statistics, both from civil and military experience, which could be cited, these may suffice:

Mathieu[26] notes that 960 cases under serum treatment gave a mortality of 38.8 per cent.; while forty cases not under serum treatment, gave a mortality of 80 per cent.

Dutertre[27] quotes Permin[28] who analyzed 330 cases of tetanus observed before the onset of the war: the general mortality was 62.1 per cent.; among those receiving serum treatment, it was 57.7 per cent.; among those receiving no serum, it was 78.9 per cent.

Other substances which are supposed to have an antitoxic effect (cholesterin, Almagia and Mendes, 1908; urea, Sewaki, 1910) have not proved themselves of value. The same may perhaps be said of phenol, according to the method of Bacelli (1893), which though employed widely and with apparent success in Italy, appears never to have given similar results in other countries. Camus (1912) showed experimentally that it had no influence on the evolution of tetanus, no matter in what amounts nor at what stage of the disease it was administered; he found that while it may possibly have some antibacterial action, it has no effect on the fixed toxin nor on the toxin in the course of fixation. Antitoxin, alone, injected simultaneously into the cerebrospinal fluid by lumbar puncture, intravenously, and subcutaneously, gave very much better results.

Exactly how the antitoxin acts on the toxin is not known. Krauss and Amiradzibi (1910), from their numerous experiments, concluded that it draws the toxin out of the cells where it is lodged, but does not itself enter into the toxin containing cells. Recovery under the use of antitoxin they believe depends on the possibility of this diffusion, and they believe that this occurs in the human body, it being unnecessary for antitoxin to be in direct contact with toxin for it to exert its neutralizing influence on the latter.

But here, again, as in the question of its prophylactic use, we must inquire as to the quantity, the site and the frequency of the injection.

1. *Quantity of Antitoxin Injected.*—No matter what the method of injection, the most important thing is to get the maximum quantity of antitoxin indicated into the patient's body as soon as possible. Delay even of a few hours may determine a fatal result; 15,000 units given within the first three hours after symptoms develop are of more value than 50,000 units given after six hours, or given in

27. Dutertre: Le tétanos et son traitement en Allemagne (1914-1915), Paris, 1915.

28. Permin: Mitt. a. d. Grenzgeb. d. Med. u. Chir. **27**:1, 1913.

divided doses. It should be made a rule to administer the total quantity indicated as nearly as may be all at one time; and after this overwhelming dose of antitoxin has been given, to keep the patient's system supplied with antitoxin, though in moderate amount, until his recovery seems assured. Dean[24] found the physiologic action of antitoxin in the blood serum could still be demonstrated as long as twenty, thirty, and even thirty-nine days after a single intravenous injection (30,000 units, U. S. A.).

As will be pointed out later, if the antitoxin is administered subcutaneously, immense quantities are indicated. For an adult, with the usual type of case, at least 100,000 units are required in the first twenty-four hours; although a less amount may be sufficient for a child or for a comparatively mild case, one cannot be sure of the fact, and it is better to give too much than not enough. Administered intravenously, a less amount is sufficient; probably 15,000 to 25,000 units should be administered at first, and if no effect is apparent, or if the good effect wears off, a similar amount should be given after the lapse of from eighteen to twenty-four hours. If injected intraspinally, from 3,000 to 10,000 units should be given, according to the weight of the patient; this injection need not, as a rule, be repeated in less than from eighteen to twenty-four hours. Even when administered intraspinally, a certain interval must elapse before the effect of the antitoxin can be apparent. Intraneural injections, rarely used at present, should be made in as great amounts as the nerves will absorb. I have injected 1,500 units into the sciatic nerve all at one time, on several occasions, and 750 units into each of the anterior crural and obturator nerves. If the injections are slowly made, all this quantity can be introduced among the nerve fibers.

2. *Site of Injection of Antitoxin.*—The following sites of injection deserve consideration: (*a*) subcutaneous; (*b*) intraneural; (c) intravenous, and (*d*) intraspinal.

(*a*) *Subcutaneous Injections.*—Thus administered, the antitoxin is absorbed by the lymphatics, transported to the veins, passes through the lungs, and finally is distributed through the arterial system to all parts of the body. Only an infinitesimal amount ultimately reaches the motor nerves through which the toxin is being carried to the spinal cord, while the greatest part is distributed to the viscera where it is of no use. This method of administration is inferior to the intravenous in the certainty and rapidity of neutralizing circulating toxin, and since overwhelming amounts are required to produce any effect it is evidently the height of extravagance so to employ it. Should it be used, at least 100,000 units, U. S. A., should be given

in the first twenty-four hours. I have cognizance of two cases in the Episcopal Hospital, the cost of the treatment of which may well be compared: In the first patient, antitoxin was administered subcutaneously in appropriate doses (99,000 units the first day; 65,000 units the second day; 60,000 units the third day), to a total amount of 224,000 units, and recovery followed. The cost to the Episcopal Hospital was about $180. Shortly before this, I had under my own care a severer case of tetanus: On the first day, 3,000 units of antitoxin were administered intraspinally; on the second day, 750 units were injected into the sciatic nerve, and 750 units were injected deeply into the tissues around the wound. The patient recovered, and the total cost to the hospital for antitoxin was about $3. The quantities of antitoxin used in each case were logically correct, but only in the second case was the mode of administration rational.

(*b*) *Intraneural Injections.*—Since the more general adoption of intraspinal injections, the intraneural method has been less used. As it is a well ascertained fact that most, possibly all, of the toxin reaches the spinal cord only by traveling up its nerves, it is theoretically logical to inject the antitoxin into the nerves in order that, like the toxin, it may not only block the nerves against further absorption but may reach the spinal cord by the easiest road. That it will reach the cord admits of no doubt (Sawamura, 1909). Accordingly, it should be injected into the nerves at the roots of the limbs.

The conclusions reached by Teale and Embleton[8] do not seem justified by the proof they offer. They assert that antitoxin does not reach the central nervous system by way of the axis cylinders or neural lymphatic channels any more than it does by the arteriovenous circulation. But the latter conclusions are based largely on their inability (1) to demonstrate by sensitization tests on susceptible animals the presence of horse serum in the spinal cord after it had been injected into the nerves, and (2) to demonstrate by inoculation of antitoxin into one sciatic nerve the physiologic manifestations of its presence in the lumbar cord of an animal into whose other sciatic nerve toxin had previously been injected. Now it seems not impossible that antitoxin, like toxin, may become fixed in the cord, while it may still be recoverable from the nerves.

But as it is manifestly impracticable to expose and inject antitoxin into all of the nerves throughout the body through which toxin is being absorbed, and as it is extremely probable even if not categorically proved that antitoxin when injected intraspinally acts on the toxin already in the nerve roots or spinal cord, it is nearly everywhere admitted that intraspinal are of more value than intraneural injections; especially valuable when the site of inoculation with tetanus bacilli is doubtful or unknown. The only methods we possess for

reaching all the nerves at once are: (1) intravenous injections, and (2) intraspinal injections. In no case, therefore, should we depend on intraneural injections alone.[29]

(*c*) *Intravenous Injections.*—The effects of these were studied experimentally by von Graff (1912), and subsequently by numerous other investigators. It is the surest and quickest way to neutralize the circulating toxin, and thus to prevent more of it from reaching the nerves and spinal cord; but it does not enable antitoxin to overtake and neutralize toxin already in the nerves or spinal cord, and it is the latter toxin, not the circulating toxin, which is doing the damage.

Teale and Embleton[5] came to the conclusion as the result of their experiments that antitoxin does not pass to the central nervous system by way of the blood vessels, but that it acts simply by combining with the circulating toxin and thus prevents its reaching the central nervous system. It is true that Dean[24] proved that antitoxin may be found in the cerebrospinal fluid in varying, but never very large, amounts, after the intravenous administration of large amounts; but if it is of any value in the cerebrospinal fluid it certainly is more rational to insert it directly by lumbar puncture, in concentrated form, than to administer it intravenously.

(*d*) *Intraspinal Injections.*—First used successfully in 1899 by von Leyden, who had no doubt that antitoxin was conveyed rapidly to the medullary cells after its injection into the subdural space of the lumbar cord, it is a method which is in danger of being neglected owing to modern experimental researches. But in spite of the large majority of experiments which at first sight tend to show that the intraspinal administration of antitoxin is of no therapeutic value, because it cannot be absorbed from the cerebrospinal fluid, there is sufficient clinical evidence on record to show that antitoxin can be and is absorbed into the nerve roots or cord directly from the cerebrospinal fluid. And even if this were not so, even if the *modus operandi* of antitoxin thus administered remained undetermined, that fact should in no wise hinder any rational physician from employing it by this route in the treatment of tetanus, since the clinical evidences of its value are overwhelming and irrefutable. Who does not know, except perhaps laboratory workers themselves, that the proper func-

29. Pratt (New York M. J. **107**:737, 1918) adopted in one case injection (15,000 units) into the vertebral artery; one artery, he asserts, delivers blood to both sides of the body. The result is not given. He points out that the circulation from this artery goes chiefly to the medulla and cord, very little to the brain. Cocain injected into the common carotid anesthetized the head and neck without medullary involvement; so he assumes that the circulation through the circle of Willis is not free in either direction.

tion of science is to explain art, not to forbid it to employ means clinically successful because so far inexplicable by laboratory researches? The facts remain, let them speak for themselves: they are both experimental and clinical, and they demonstrate beyond the shadow of a doubt that it is absolutely unjustifiable to neglect this route of administration of antitoxin in the treatment of tetanus. In addition to the now classical experimental work of Park and Nicoll [32] we have the later researches by Sherrington, presented by Bruce [33] and by Andrewes; [34] as well as those of Golla [35]—all of which demonstrate that if administered intraspinally antitoxin is effective. *How* it acts, may not yet be certainly known; let the laboratory workers continue their experiments until they find this out; that is their true function; but let them not dare to forbid its employment because its means of action remain unknown: *ne sutor supra crepidam.* It is clinical work which here, as elsewhere in medicine, leads the way by Art, leaving the *how* and the *why* to be explained if possible by Science.

The clinical results of the intraspinal use of antitoxin are shown in Table 1.

TABLE 1.—Results of Treatment by Antitoxin Intraspinally

Author	Patients			Mortality, per Cent.
	Number	Recovered	Died	
Luckett, 1904	4	4	0	0.0
Rogers, 1905	7	4	3	43.0
Hofmann, 1907	16	14	2	12.5
Permin, 1914	28	11	17	60.7
Nicoll, 1915 (collected cases)	20	16	4	20.0
Gibson, 1916	4	4	0	0.0
Ashhurst, 1920	15	10*	5	33.0

* One patient died subsequently of pneumonia.

It is true that in most of these patients antitoxin was given also by other routes besides the intraspinal (i. e., intravenously and subcutaneously), and that other proper methods of treatment were not neglected (use of sedatives, careful nursing, etc.); for tetanus is a terrible disease and must be fought with every available weapon. It must also, however, be borne in mind that the list given in Table 1 includes (at least in the case of my statistics) patients to whom antitoxin was given intraspinally so late that it is scarcely fair to

32. Park, W. H., and Nicoll, M., Jr.: Experiments on the Curative Value of the Intraspinal Administration of Tetanus Antitoxn, J. A. M. A. **63**:235 (July 18) 1914.

33. Bruce, David: Lancet **1**:680 (May 5) 1917.

34. Andrewes, F. W.: Lancet **1**:682 (May 5) 1917.

35. Golla: Lancet **1**:686, 1917.

include them in seeking to determine the value of this avenue of administration. If treatment is both efficient and early the mortality from acute tetanus probably should not exceed 20 per cent.

3. *Frequency of the Injections.*—If the rule I have already enunciated be followed, namely, to administer the total quantity indicated as nearly as possible all at one time, and particularly if the intraspinal (3,000 to 15,000 units) and intravenous (20,000 to 30,000 units) methods are employed, the injections will not need to be repeated very frequently. Intraspinal injections usually are to be repeated from every twenty-four to thirty-six hours unless improvement commences; the intravenous injection need not be repeated for several days if improvement commences, but if the patient continues to get worse, and certainly if a less amount than 20,000 units has been injected at first, this amount should be repeated within from twenty-four to thirty-six hours.

4. *Technic of the Injections.*—Finally, a word may be said as to the actual technic of these injections. In giving intraspinal injections for which narcosis (preferably by chloroform) usually is required, from 5 to 10 c.c. of spinal fluid should first be withdrawn. Then the antitoxin is slowly injected, undiluted. I have seen no harm arise from the injection of the undiluted serum, and I believe it is desirable to have it present in the cerebrospinal fluid in as concentrated form as possible. It is true that at subsequent punctures on the same patient one often finds the cerebrospinal fluid turbid or cloudy (an evidence of amicrobic meningitis), but this seems to do the patient no harm. In some cases, also, I have seen delirium follow the injection; but as the patients recovered this must be regarded as a negligible complication.

For intravenous injections it is customary to dilute the serum with saline solution up to a total quantity of 500 c.c. or thereabouts; and I am convinced that the injection of the undiluted serum intravenously would be injudicious because of the likelihood of producing thrombosis and perhaps embolism.

C. *Depression of Function of Spinal Cord.*—The third indication in the treatment of tetanus is to depress the functions of the spinal cord. This is equally important as the effort to eliminate the supply of toxin, and with those to neutralize the toxin already formed, because in almost every case there is a large amount of toxin which has become impregnably entrenched in the central nervous system, particularly in the spinal cord, and none of the methods of treatment hitherto discussed has any influence over it. Until its action is exhausted it continues to stimulate the motor, and, to a less degree the sensory, tracts of the spinal cord, and kills the patient by exhaustion.

We have at our disposal a number of drugs whose main therapeutic action is to render the spinal cord less susceptible to stimulus; and administration of one or more of these remedies forms an integral part of any rational plan for the treatment of tetanus. The drugs most often employed are chloral, chlorbutanol, and similar products; the bromids; magnesium sulphate; and of late years sodium persulphate. These drugs are to be administered until the therapeutic effect which is desired has been obtained. Ordinary doses are not sufficient, but it is quite possible to kill the patient by an overdose. In nine patients in my own series of cases (1912) the condition at death was noted: Only three patients died in spasm or convulsion, while six died in complete relaxation; and in some of these cases the condition was due to overdoses of the spinal depressants employed. Especially dangerous, I believe, is magnesium sulphate, which, as Bérard and Lumière [30] express it, is efficient only in *doses paramortelles*. I agree with them also that chloral, in doses of 4 to 10 gm. daily, is the most efficient and inoffensive of spinal depressants; and I habitually employ it in conjunction with the bromids.

Their own experience [31] with intravenous injections of sodium persulphate is worthy of great attention. Lumière employed it in eight cases before the war: only two of the patients died. During the war, he states that of twenty-nine patients treated efficiently by this method, only thirteen died, a mortality of 45 per cent. He injects intravenously once or twice daily 20 c.c. of a 5 per cent. solution of the drug, which should be pure, and neutral in reaction. These injections are to be repeated daily or less often for the first or second week of the disease. But as Bérard and Lumière [30] themselves acknowledge, it is not so efficient in very acute cases, and chloral should be used also. They report a total of 116 cases which they treated during the war, with fifty-eight deaths, a mortality of 50 per cent. But it must be remembered that many of the war cases of tetanus were late, chronic, local or recurrent cases, and so the mortality cannot well be compared with that of the cases seen in civil life. Sodium persulphate should be preserved in solid form (dry!) in 5 gm. quantities, in sealed tubes. The solutions can be preserved only a few days (not more than eight) and are decomposed by heat. They must be prepared fresh by dissolving the contents of the 5 gm. tube in 100 c.c. of cold sterile distilled water. The solution corrodes metals, and metal syringes and needles must be very carefully washed after use. After the injection the clonic spasms cease, but the tonic spasm is unaffected. Vomiting occurs in about half the cases after the injection. He regards this remedy as antitoxic as well as depressant, and seems not to have employed any antitoxin in addition. Other authorities, however, doubt its antitoxic power.

30. Bérard and Lumière: Presse méd. **26**:469 (Sept. 12) 1918.
31. Lumière: Lyon chir. **12**:411, 1915.

TABLE 2.—Cases of Tetanus Under the Author's Care

No.	Date of Admission	Age and Sex	Site of Wound	Date and Nature of Injury	Incubation Time from Injury to Symptoms	Time from Symptoms to Institution of Efficient Treatment	Type of Case	Summary of Treatment		Result
								Original Injury	Tetanus	
1	9/28/08	34, M.	Abdomen and thigh	Sept. 24, 1908; brush burns, hematoma; thrown from wagon and dragged	14 days	1 hour	Slow in onset, but severe convulsions	Ordinary antiseptic	Sedatives; antitoxin subcutaneously (129,000 units) and intraspinally (3,000 units); total antitoxin, 132,000 units in 10 days	Recovered Oct. 31, 1908
2	9/23/08	55, M.	Cranium	Sept. 23, 1908; gunshot fracture of temporal region and rupture of eyeball	4 weeks	12 hours	Mild, slow in onset; no convulsions	Aseptic; no operation	Sedatives; antitoxin subcutaneously 31,500 units in 8 days	Recovered; eyeball enucleated Nov. 17, 1908
3	11/12/08	49, M.	Foot	Nov. 4, 1908; punctured wound by rusty nail	8 days	24 hours	Medium severity; no convulsions	Home treatment by ham fat, etc.	Sedatives; antitoxin intraspinally (3,000 units) and later subcutaneously (27,000 units); total amount, 30,000 units in 6 days	Recovered Nov. 18, 1908
4	12/19/08	19, M.	Foot	Nov. 30, 1908; punctured wound by splinter from mill floor	18 days	24 hours	Severe convulsions before admission	Wound dressed twice but patient never returned for further dressings	Three splinters removed from wound; sedatives; antitoxin intraspinally, 3,000; intraneurally, 750 units, and into muscles around the wound, 750 units; total in 2 days, 4,500 units	Recovered Dec. 31, 1908
5	10/16/09	63, M.	Fingers	Sept. 28, 1909; lacerations by machinery	18 days	24 hours	Severe no convulsions	Ordinary antiseptic dressings	Fingers amputated; sedatives (chlorbutanol; antitoxin intraspinally 1,500 units	Died, Oct. 19, 1909, after being comatose 24 hours
6	11/20/09	11, F.	Knee	Nov. 11, 1909; abrasion from fall	9 days	40 hours	Slow in onset, severe convulsions	Bread poultice at home	Sedatives (chlorbutanol); antitoxin intraneurally 3 times, total 9,000 units; intraspinally 1,500 units; subcutaneously, 3,500 units; total antitoxin, 13,500 units in 3 days	Died, Nov. 22, 1909, after being comatose 3 hours
7	2/ 4/10	45, F.	Toes	Jan. 23, 1910; laceration; fall downstairs in bare feet	12 days	Few	Mild, slow onset, no convulsions	Neglected for 10 days, then iodin	Sedatives; antitoxin subcutaneously, 6,000 units in 3 days	Recovered Feb. 19, 1910
8	2/21/10	23, F.	Scalp	Jan. 26, 1910; lacerted wound; hit by brick	19 days	1 week	Slow in onset, severe, no convulsions	Sutured, no drain	Abscess opened; sedatives; antitoxin intraspinally, 3,000 units; subcutaneously, 21,500 units; total antitoxin, 24,500 units in 5 days	Recovered Mar. 8, 1910
9	10/ 3/10	6, F.	Arm	Sept. 7, 1910; vaccination	Unknown less than 24 days	3 days	Slow onset, very severe, 2 convulsions every hour	Treated at home by mother	Sedatives; antitoxin intraspinally, 10,000 units (Oct. 5); subcutaneously, 45,000 units; total antitoxin, 55,000 units in 2 days	Died, Oct. 5, 1910, in convulsion

10	2/ 4/11	6, M.	Hand	Dec. 23, 1910; Laceration; fall on icy street	9 days	4 days	Severe convulsions	Sutured	Sedative and magn. sulph. intraspinally; antitoxin intraspinally, 9,500 units; subcutaneously, 10,000 units; total, 19,500 units in 2 days	Died, Feb. 7, 1911, in perfect relaxation
11	11/20/13	23, M.	Sole of right foot	Nov. 12, 1913; puncture by rusty nail	7 days	9 days	Trismus first sign, convulsions after admission	Untreated	Sedatives; antitoxin subcutaneously, 18,500 in first 24 hours; intraspinally, 10,000 2d day; 9,000, 5th day; intravenously, 18,000 2d day; intravenously, 5,000 5th day	Died Nov. 25, 1913
12	10/29/15	17, F.	Finger	Oct. 15, 1915; puncture by machine needle in woolen mill	13 days	24 hours	Slow onset, local pains for 5 days, stiff neck for 1 day before admission	After 24 hours opened, no antitoxin	Healed wound opened and cleaned; sedatives, large doses; antitoxin intraspinally, 10,000 units; intravenously, 18,000 units; intramuscularly, 6,000 units	Recovered Nov. 10, 1915
13*	12/17/12	27, M.	Right finger	Before Dec. 1, 1912; crushed	9 days	9 days	Subacute onset, trismus, epigastric pain, stiff legs, spasm 3 days before treatment	Bandaged by patient	Sedatives, very large doses; antitoxin intraspinally, 8,000 units; intravenously, 28,500 units	Recovered Dec. 21, 1912; died Dec. 22, 1912 of pneumonia
14*	4/11/14	58, M.	Nose	April 7, 1914; struck by piece of bone in fertilizing plant	3 days	1 day	Severe, facial paralysis	Abscess formed and this was opened 4th day	Sedatives; antitoxin intraspinally, 18,000 units in 2 days; intravenously, 13,500 in 2 days; into facial nerve, 1,500 on 2d day	Died, April 13, 1915, in coma
15*	11/17/14	18, M.	Hand	Three weeks ago; scratch by wire nail	Over 2 weeks	5 days	Slow onset with stiffness and pains in legs, trismus, spasms, opisthotonos and risus sardonicus on admission	Untreated	Sedatives, large doses; antitoxin intraspinally, 6,000; intravenously, 15,000; subcutaneously, 3,000 units	Recovered Dec. 15, 1914
16*	10/20/15	34, F.	Right knee	Oct. 4, 1915; chafed in riding horseback	12 days	4 days	Subacute onset, pains in side, back muscles and neck, opisthotonos before treatment and constant spasms	Ointment applied by patient	Sedatives, very large doses; antitoxin intraspinally, 9,000 units; subcutaneously, 20,000 units in 6 days	Recovered Oct. 20, 1915
17	10/26/16	32, M.	Foot	Nov. 16, 1916; puncture by rusty nail	8 days	2 days	Slow onset with stiffness of cheeks, jaw and neck	Untreated	Sedatives; intraspinally, 10,000 units; intravenously, 27,000 units in first 18 hours after admission	Recovered Dec. 13, 1916
18	9/18/20	13, M.	Feet?	?	?	5 days	Slow onset, colonic spasms on admission and opisthotonos	Untreated	Sedatives; intraspinally, 10,000 units; intravenously, 35,000 units in first 12 hours after admission	Recovered Oct. 1, 1920

* Cases 13, 14, 15, 16 seen in consultation; all the others treated in the Episcopal Hospital, Philadelphia.

Spinal anesthesia is suggested by Bérard and Lumière and in one patient they secured marvellous relief from respiratory cramps by injecting a local anesthetic into both phrenic nerves at the root of the neck.

D. The patient, as well as the disease, must be treated; but it is perhaps unnecessary here to dwell further on nursing, feeding, and meeting every untoward symptom as it arises.

I have had under my personal observation eighteen cases of tetanus, all in civil life; seven of these patients died (38.8 per cent.), one of them (Case 13 in Table 2) from pneumonia, after apparent recovery from the tetanus. In three other fatal cases, treatment was neither early nor efficient enough to be successful (Cases 6, 9 and 11); while the death of one patient (Case 10) was clearly due to an overdose of magnesium sulphate intraspinally. This leaves thirteen cases in which treatment may be regarded as early and efficient; among these, only two patients died, a mortality of 15.38 per cent.

D. *Summary.*—My views as to the treatment of tetanus may be summarized by narrating the history of the next to the last patient to come under my care: he was treated early and efficiently, and recovered.

Case 17.—*History.*—M. H. J., aged 32, a member of one of the faculties in the University of Pennsylvania, referred to me by Dr. Alfred Stengel, was seen in my office about 7 p. m., Nov. 26, 1916. He stated that two days before (Nov. 24, 1916) he noted in the morning that his cheeks felt stiff, as if he had been out in the wind all day (which he had not). The next day he felt the same way, and on the morning of the 26th he noticed in addition to the sensation of stiffness in the cheeks and jaw, that he was stiff in the neck and shoulders, as if from overexercise. Only then did he recollect that on November 16 or 17, while doing some carpenter work in his garden, he had stepped on a rusty nail, which had perforated his shoe and penetrated his foot from 2.5 to 3 cm. He had extracted the nail, squeezed out a few drops of blood, and the puncture had given no further trouble. He had forgotten about it until the persistence of stiffness in the jaws and neck made him apprehensive of the development of tetanus.

Examination.—This was negative, except for slight stiffness in the jaws, but the mouth could be opened well. Nevertheless, the sensation of stiffness was pronounced in the jaws, neck and shoulders.

Clinical Course.—I took him with me at once to the Episcopal Hospital; and as he rode beside me in the car I felt anxious lest he should suddenly grow worse and have a convulsion. There seemed no doubt about the diagnosis: if delay were allowed, the treatment, no matter how energetic, would come too late to be efficient.

By 8 p. m. (one hour after he came to my notice), this patient had received, under local anesthesia, 10,000 units of concentrated antitoxin intraspinally by lumbar puncture. (The cerebrospinal fluid withdrawn was reported by Dr. C. Y. White as containing no cells; and when injected into a mouse, it had no deleterious effect.) The puncture in his foot could not be certainly identified, and nothing was done to it. He received also, by mouth, chloral hydrate,

1 gm., and sodium bromid, 2 gm. By 12 midnight his temperature had risen to 39 C. (102.2 F.) but had fallen again to 37.8 C. (100 F.) by morning.

Nov. 27, 1916: The patient was delirious, muttered under his breath, and was so restless that restraint was necessary. His jaws were firmly locked, but with effort could be opened slightly. The neck was stiff, but there was no opisthotonos. Knee jerks were increased on both sides, there was a slight Babinski reflex on the left side. He was given 27,000 units of antitoxin (diluted in saline solution) intravenously, and following this, he had a severe chill and his temperature rose to 40 C. (104 F.). Immediately thereafter it fell to 37 C. (98.6 F.) and never rose subsequently above 37.8 C. (100 F.). He was given chloral hydrate, 2 gm., and sodium bromid, 4 gm., every three hours by rectum, as he could not swallow. About 6 p. m., the patient began to clear up mentally. His neck continued very stiff. At 7:30 p. m. he was given two drops of oleum tiglii (Croton oil). He voided 830 c.c. of urine during the day.

Nov. 28, 1916: The patient was improving; the knee jerks were increased; there was a slight Babinski reflex on both sides. The head could be flexed beyond the plane of the body, the neck being quite rigid. There was some headache. The bowels were opened twice. He voided 800 c.c. of urine. Chloral and bromid were given as on the preceding day.

Nov. 29, 1916: He lay on his side curled up, and slept most of the time. He took nourishment well. There was some headache. The neck was slightly stiff still. Chloral and bromids were given only in one third the former dose, and by mouth.

Dec. 1, 1916: Urticarial rash appeared over the whole body. Itching was relieved by phenolated petrolatum.

Dec. 2, 1916: Urticarial rash was more pronounced. This was relieved by 5 drops of pituitary extract. He was nauseated, but was relieved by vomiting.

Dec. 13, 1916: Convalescence was uneventful. He went home today.

RIB GRAFTING OPERATIONS FOR THE REPAIR OF BONE DEFECTS AND THEIR END-RESULTS

AT LETTERMAN GENERAL HOSPITAL *

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Of late it seems as though a few definite conclusions may have been reached as to the growth of bone. Gallie and Robertson,[1] Hey-Groves [2] and others have presented the results of many years of research in scores of laboratories and have outlined the theories deduced in admirably clear papers.

Opinions as to the proper way of performing a bone grafting operation still seem, however, to be diametrically opposed. In this country we are led by Albee's energetic advocacy of the massive transplant. The French, as far as I can judge, follow Delagenière's method of thin, periosteo-osteal flaps. The English, if I interpret Sir Robert Jones' expressions of opinion correctly, regard the bone graft with considerable distrust and put their faith in shortening and suturing ununited fractures. In order, then, to arrive at an estimate of the value of bone grafts, and at a conclusion regarding the proper method of performing them, it would seem worth while for each man to gather his results and review them as honestly as possible. It might be even fairer to appoint an impartial commission—if there is such a thing—to conduct the inquiry.

I wish to present a series of cases in which one particular method of grafting was employed. The series is small, but I have tried to judge the results as impartially as possible. I have tried to follow the patients, soldiers at Letterman General Hospital, after their return to civil life and to find out, as best I could, the functional value of the grafted bone. In presenting the series I am making no plea for the method I have followed.

It has not been easy to arrive at a just basis for judgment. Roentgen rays give objective and impersonal evidence, but they are

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* Read before the Section on Orthopedic Surgery at the Seventy-First Annual Session of the American Medical Association, New Orleans, April, 1920.

1. Gallie, W. E., and Robertson, D. E.: The Repair of Bone, Brit. J. Surg. **7**:211 (Oct.) 1919.

2. Hey-Groves, E. W.: Methods and Results of Transplantation of Bone in the Repair of Defects Caused by Injury or Disease, Brit. J. Surg. **5**:185 (Oct.) 1917.

notoriously fallacious guides to function. On the other hand, wage-earning capacity and even the extremities themselves are often faulty indexes of the value of a graft. Bony defects resulting from battle wounds are so frequently accompanied by grave nerve lesions and extensive loss of muscles and tendons that the extremity remains quite useless, even with the most perfectly repaired bone.

It might be best to eliminate from the statistics of bone grafting operations all cases in which there were grave nerve lesions or extensive injuries of the soft parts. This, however, would reduce the series by two-thirds or more. The better plan will be, I suppose, to combine roentgen-ray evidence with estimates of the actual functional value of the extremity.

The rib was chosen as a source of supply for the graft for several reasons, theoretical and practical.

THEORETICAL ADVANTAGES OF RIB GRAFT

In theory it was argued that most, if not quite all, of the osseous part of the graft dies, and that only its osteoblastic elements (both the epiosteal osteoblasts and the osteoblasts lying at the mouths of open haversian canals) survive. It was argued that experiments have shown that the more accessible these viable osteoblastic elements of the graft are to the nutrient juices of the body, the sooner does their proliferation begin; furthermore, it was thought that of the transplanted osseous tissue only those minute portions which lay immediately under the periosteum, and along the endosteum of the cancellous portions, survive, i. e., again those portions which are most readily accessible to the nutrient juices. If this be true, the viability of the graft would be in inverse proportion to its mass. Thick, poorly permeable pieces of bone die off more slowly, and the processes of absorption and restitution are more tardy; more permeable pieces, of greater surface area and less cubic content, are attacked and restored more quickly. It was, therefore, reasoned that a graft with a thin, vascular, soft cortex and a large proportion of cancellous marrow spaces would be better than one with a thick, dense cortex and little or no cancellous tissue. It was thought that the inevitable absorption of osseous tissue would take place more quickly in the thin graft than in the thick one, that deposits of permanent bone would be laid down more rapidly and that the graft might be sooner put to use and submitted to the forces which cause active functional hypertrophy.

Whether or not these theoretical premises have been borne out by practice I am not prepared to say. I am inclined to doubt it.

Gallie had not published the outcome of his investigations at the time these operations were undertaken. His observations seem to

show that the processes of absorption outstrip those of replacement. He says that after the lapse of five weeks the graft is exceedingly porous and fragile, and that it remains so until the irritation from the presence of dead bone disappears, i. e., after eight or nine weeks. He states that the longer the gap, the thicker the graft used to fill it should be, so that it may retain strength during the stage of osteoporosis and that, although a porous bone graft retains more cellular activity than a massive one, it has also a much greater tendency to rapid absorption and must be used with care. Hence, he warns against the use of cancellous bone, such as the rib, when the graft is to be put under strain, and prefers a strong piece of tibia surrounded by a number of smaller strips, the latter to facilitate the access of an increased number of osteoblasts.

The series under consideration would tend to show that Gallie is right, that the very tardiness with which the more massive grafts are absorbed may be of advantage, and that they offer firm mechanical support for a longer period. Once the invading capillaries have started their work of absorption and replacement, there may be more new bone laid down in the dense substance of a massive graft than in the frailer body of a thin one, which is probably so rapidly swept away that its trellis is gone by the time the osteoblasts are ready to lay their bony deposits on it. I shall refer to this question again in considering the frequency of fractures of grafts. Clinical observation must settle this point; laboratory experiments can scarcely help us.

The rib, then, was chosen on theoretical grounds in preference to the tibia because it was softer and more easily permeable. It seemed that more of it might live and that what was doomed to die would be absorbed and replaced more quickly. It was chosen in preference to the osteoperiosteal transplant of Delagenière—a free graft of periosteal membrane with a few bone chips clinging to its under surface—because it was not so utterly flimsy and because, while it was not so strong as a tibial graft, it had some substance and some strength. It was, therefore, not necessary to place entire reliance on casts and other external means of fixation and support.

PRACTICAL ADVANTAGES OF RIB GRAFTS

Aside from the theoretical, the rib graft offers a number of practical advantages. No special instrumentarium is required for the operation. This is of minor importance. Letterman General Hospital is equipped with the Albee outfit, and so are most institutions where bone grafting operations are performed. At any rate, if they are not properly equipped, they should be. However, the Albee outfit does break down now and then, often at the particular moment when

it is most needed, and it is a reassuring feeling, when undertaking an operation, to know that one is not entirely at the mercy of an electric saw.

The rib is much more workable than the tibia. It can be bent or cut into the desired shape with bone-cutting forceps or a stout pair of scissors. It can be pierced with a stout needle. It has more spring and is not so liable to crack as the tibia. And in spite of its lightness, it is quite strong; strong enough to give good support to the lighter bones, especially the radius and ulna. Its strength lies in its curved cross-section, just as weight for weight a tube is stronger than a solid cylinder, or a piece of channel iron is stronger than a sheet of the same metal. When the rib is split in halves longitudinally, it forms a stout trough that can be wrapped about half the circumference of one of the smaller bones so as to hold it firmly in position. Again, the rib can be split into staves, with which two fragments may be firmly surrounded and effectually splinted. It makes an excellent, pliable, workable internal splinting material.

The supply is practically unlimited. To take 6 inches or so from one or two of the ribs does not affect the well-being of the patient.

The operation does not entail a lengthy stay in bed, nor does it keep the patient off his feet for more than a few days, a week at most. Many patients on whom bone grafting operations are indicated have been so long bedridden that they dread the prospect of an additional six or eight weeks in bed on account of a weak tibia.

The operation itself is neither more difficult nor more dangerous, nor does it take longer to resect a piece of rib than to saw a piece out of the tibia.

The sterility of the operative field is always more or less a matter of doubt in bone defects due to war injuries. In these dubiously aseptic cases, the less massive and more viable rib graft has a greater chance of weathering an infection than a heavy tibial graft. The graft acts for the first few days much like a foreign body. The less dead material one implants the less the risk of lighting up dormant infections. The rib, therefore, has many practical advantages over the tibia.

I am quite willing, however, to concede that all of these advantages may be more than offset if statistics of end-results show that the graft from the rib is not as good as that from the tibia.

TECHNIC OF RIB GRAFTING

The technic of rib grafting is not difficult. The bed is prepared as for any other graft. It must contain no sequestrums buried in dormant pus. The wound should be healed, if possible. The scar

should be reactionless and able to withstand heavy massage without breaking down. If it is adherent to the bone it should be excised, preferably at a preliminary operation, and the site prepared for the graft should be covered by healthy skin. If it is not possible to mobilize the wound edges sufficiently to cover the bone with nonadherent skin, sound skin and subcutaneous fat should be brought down from elsewhere, either as a pedunculated flap or as an Italian graft.

The field should be prepared antiseptically and aseptically forty-eight hours before operation.

The extremity should be made bloodless with a tourniquet and the bones exposed through a generous incision. Sclerosed and eburnated bone should be removed from the ends of the fragments and the fragments brought into proper line by traction and by the excision of distorting scars if necessary. A hole should be bored through the end of each fragment, and a stout kangaroo tendon threaded through it. A hot compress is placed in the wound, the tourniquet removed, and bleeding stopped. One bloodsoaked compress should be saved to wrap the graft in. The patient's own blood is probably the best and certainly the most natural medium in which to keep the graft. An incision is made over the fifth or sixth rib, beginning at the costicartilaginous junction, i. e., about half way between the nipple and sternum and extending backward far enough to expose the required amount of bone. The front part of the fifth or sixth rib is usually chosen because it is straightest. For a curved graft, to be placed in the jaw or skull, the surgeon should select a lower rib, which is more curved, and take the piece farther back. In women, the sixth or seventh rib may be used so that the incision may follow the lower border of the breast. The breast may be lifted up and a piece of rib taken from beneath it. Grafts for the repair of nasal defects may be taken from the costicartilaginous junction, including both bone and cartilage.

The one difficult step in the operation is getting the rib out together with the periosteum without opening the pleura. Entering the chest does no serious harm, but it had better be avoided. The pleura was opened seven times in this series without noticeable effect except in one patient in whom a subcutaneous emphysema developed which persisted a few days. If the chest is opened, the opening should be stopped with a large moist compress while the rib is being resected, so as to prevent the lung from flapping about in the gaping pneumothorax.

A sufficient length of rib is exposed, and the muscle, the serratus magnus, retracted. The attachment of the external intercostal muscle is severed with a knife. The muscle fibers slant from above and behind downward and forward. The knife should therefore pass forward along the upper edge of the rib and backward along the

lower edge. If passed in this direction, it remains in the angle made by the muscle fibers and the rib. The rib should first be cleared of its muscular attachments for an inch or two in front. The knife should be used very carefully and delicately until the pleura comes to view and the speckled tissue of the lung is seen sliding back and forth under it. After an inch or two of the rib is clear the pleura may be very gently pushed away from it with the handle of the knife or some other blunt instrument. The rib must be freed from the intercostal muscles with a knife. Blunt dissection, either with gauze or with an instrument, will not separate the costal periosteum from the pleura. A healthy pleura is as thin as tissue paper. It will inevitably tear if a blunt instrument is used in an attempt to strip the periosteum of the rib from it. The minutest hole is irreparable. Once punctured, the pleura always tears farther. If, however, the intercostal muscles are cut away from the rib for an inch or two in the manner described, they may be gently pushed off the pleura and the pleura separated from behind the rib. When the inner surface of the rib is free, curved rib shears may be inserted and the front end of the graft cut through. If the rib is then forcibly retracted outward, it is easy to cut away the intercostal muscular attachments as far back as is necessary. The pleura drops away from the rib by its own tension so that there is little danger of entering the chest.

Six or more inches of bone may be obtained without difficulty.

There is no danger of wounding the intercostal artery if the front of the rib is cleared first and if the knife hugs the bone closely. The artery comes out from under the edge of the rib between the two axillary lines. If it is exposed before the axilla is reached, it is easily avoided. If it is wounded, bleeding should be stopped by pressure until the rib is removed. Blind attempts at grasping the artery with a hemostat endanger the pleura. The artery should, however, be exposed and securely tied before suturing the wound, especially if the pleura is open, so that there may not be bleeding into the pleural cavity.

No attempt should be made to suture the pleura separately if it has been opened. The hole should be stopped with a compress while the muscles are being sutured together. It is well to take the last stitch during expiration so as to get as much air out of the chest as possible before closing it.

The wound in the chest is closed by an assistant while the operator prepares the graft. He drills a hole for the kangaroo tendon suture through each end of the excised piece of rib. If the ends of the fracture are to be separated, the distance between the holes in the graft should exceed the distance between the holes through the fragments. After the holes have been drilled, the lower edge of the graft is split longitudinally with an osteotome or a stout knife and the rib opened

out like an oyster, a hinge of periosteum remaining along the upper border. The rib is pliable enough to be straightened out somewhat with the hand if it is too curved, although it tends to spring back into its original shape. The kangaroo tendon sutures are threaded through the holes in the graft and the fracture ends laid into the trough formed by the split rib. Both ends of each suture are passed under the bone with a ligature carrier and the suture is firmly tied about the whole graft and the bone. If the fragments tend to move in the trough, a second ligature may be passed either above or below the first. This may either be led through drill holes or simply wrapped about both graft and

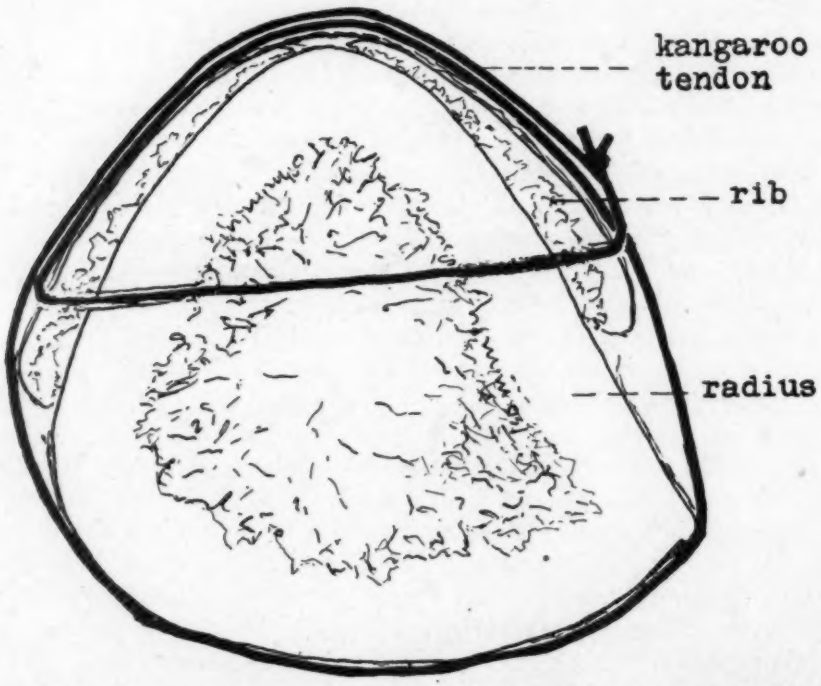


Fig. 1.—Cross-section of radius and rib graft. The rib is split longitudinally, its two halves are joined above by a periosteal hinge. Kangaroo tendon passes through drill holes and encircles both rib and radius.

fragment. The periosteum of the bone is sutured as well as may be about the rib, the soft parts are brought together accurately, the wound is closed, and the extremity is encased in plaster.

Variations applicable to different types of cases will suggest themselves. In a large defect of the humerus, two ribs were taken, split in halves, and the fragments wrapped about the two ends of the humerus like barrel staves. The space between the staves was sprinkled with iodoform powder and left to fill with blood-clot. An

artificial marrow cavity was made. The result was unsuccessful. The defect was bridged and the graft united, but the arm was subsequently refractured. There is still nonunion. The arm is not nearly so flail as it was. There is some power; but the union is by no means firm (Fig. 27).

The rib graft is particularly useful when sepsis makes the success of a more massive transplant unlikely. In one patient, with a tibial defect, a rib graft was used successfully while the wound was still open. There was a large scar over the front of the shin, extending down through the defect, and in its middle a granulating area about half a centimeter in diameter. An Italian plastic operation to cover the defect was thought inadvisable because of recent attacks of streptococcic infection. An attempt to unite the fibula (which was intact) with the tibial fragments, a procedure which entails considerable manipulative trauma, was thought inadvisable for the same reason, and I was loath to risk the stability of the one remaining bone of the leg on so slender a prospect of success. An incision was therefore made on the inner posterior side of the leg, the side farthest from the scar, and the back of the tibia exposed without disturbing much of the fibrous tissue lying between the bone ends. A piece of rib 5 inches long was split in two longitudinally and each half laid against the back of the tibia, spanning the defect. The wound was closed loosely and the leg put in a splint. The man had some fever following the operation, but none of the graft came out, the fracture united, and the man can support his whole weight on the leg, although he still wears a brace for a foot-drop due to destruction of the extensor muscles (Fig. 22).

The rib is particularly well adapted for filling in cranial defects. The convexity fits in well with the curve of the cranium and gives an excellent cosmetic result.

I think that I have erred in the after-treatment of these patients in not fixing their limbs sufficiently firmly or sufficiently long. A number of them were fixed in splints instead of in circular casts. Most of the casts were removed too soon. Had I to do them over again I should, in the light of Gallie and Robertson's experiments, apply circular plaster dressings and leave these on at least two months.

The question when to take off the cast, when to leave off all support, and when to begin active motion and use, should be determined by information as to the time when absorption of the transplant ceases, when hypertrophy begins, and when the bone reaches its maximum size—not density, but size. Gallie's experiments show that absorption is complete at the end of two months. Fixation, therefore, should somewhat exceed this period, and plaster should be used

for ten or twelve weeks. Rib grafts take several months before they begin to show roentgenographic evidences of growth; but that they finally do grow and become stronger may be seen in a number of roentgenograms.

END-RESULTS

I have tried to follow as many of the patients as I could up to and after their discharge, and I have tried to judge honestly of their condition. As I said in the beginning, I scarcely know what to call a success and what a failure. Is the operation a success in a man with a radius which is firm clinically and according to roentgen-ray findings, but with a median and ulnar palsy and the remains of an ischemic contracture, with a stiff claw hand? And is the operation on the radius of a man whose roentgenogram reveals a fractured graft, but who can hold a chair out at arm's length, a failure? I shall call those operations failures which result clinically in nonunion and flail extremities; I shall call those operations partial successes when there is roentgenographic evidence of refracture or bone absorption, but in which the extremities are useful clinically. I shall call those operations successes in which both roentgen-ray and clinical observation indicate firm union.

The series includes twenty-two patients operated on at Letterman General Hospital from January to October, 1919. One attempt at replacing the carpals in a flail wrist by a free rib graft was accompanied by suppuration and was a failure. There are ten defects of the radius alone, seven successes and three partial successes. There is one defect of the ulna alone, a partial success, refractured at operation. There are five defects of both radius and ulna, one partial success and four successes. There are three defects of the humerus, all failures. In the man shown in Figure 27 the large defect is repaired, but there are a refracture and nonunion. There is one defect of the tibia and one of the jaw, both of which were successfully repaired. Eleven of the men had more or less suppuration following operation; in some of them the suppuration came on late, sometimes a number of weeks after operation. In but one case what apparently was the entire graft was lost. This occurred in a fracture of the humerus. Others healed spontaneously without sequestration.

Refracture occurred in seven, almost one third, of the twenty-two cases.[3] The graft always broke near the middle, the ends being united to the matrix bone. That these fractures occurred at the middle of the grafts while the ends remained firmly welded to the ends of the main fragments shows the truth of Gallie's statements that the proc-

3. Figures 4, 14, 15, 18B, 26 and 28. Compare with accompanying table.

esses of absorption outstrip those of replacement, and that the ingrowth of bone occurs largely through the migration of bone cells from the neighboring bony bed rather than by proliferation of the osteogenic tissue of the graft itself.

The ends of the graft, those nearest the source of living bone supply, showed no atrophy; they remained firm and dense. It was the middle of the graft, the part most distant from the bone of the host, that was thinnest and that gave way most easily under strain. Most of these refractures have remained ununited so far as I have been able to follow them. In two of them, however, the refracture united, in one with considerable callus. These cases, a parallel to those cited by Hey-Groves,[2] prove that the grafts were alive (or had been supplanted by living bone cells) and that their bone had powers of osteogenesis (Figs. 23 *B*, 23 *C* and 24). It is true that two of the seven were firm but were refractured after operations in which much bone was removed. It is also true that most of the fractured ones were not splinted with a view to obtaining reunion. Still, refracture of the graft occurs so frequently that it makes me look askance at the rib graft for the repair of defects that put the implanted bone under much strain.

CONCLUSIONS

1. The rib graft is a feasible procedure.

2. The viability of the grafts is great, even in the presence of suppuration.

3. They are more apt to survive in the presence of infection than more massive grafts.

4. They are rapidly absorbed.

5. They hypertrophy slowly.

6. They are prone to refracture (seven out of twenty-two cases).

7. They are not so good as tibial grafts for the repair of large defects or when the graft is to be put under strain.

8. They are particularly useful when no great demands are made on the strength of the bone, in repairing defects of the skull and in facial plastics. In such cases they should be used by preference.

9. Of a series of twenty-two cases, three were failures, thirteen were successes and six were partial successes.

REPORT OF CASES

CASE 1.—A. O., Pvt., Co. G, 18th Inf., aged 28. High explosive wound of left wrist, May 4, 1917. Entered Letterman General Hospital Sept. 26, 1918. Flail left wrist. Distal end of radius and ulna, and all carpal bones, except trapezium are missing. Hand deviates to ulnar side.

Feb. 3, 1919: Rib graft to take place of missing carpal bones and to correct ulnar deviation of hand. Graft inserted into a newly formed joint cavity with a true synovial lining. Periosteum scraped off one side of one of the grafts, and distal end of radius freshened to bring graft into connection with it. Wound closed. Splint applied. Pleural cavity was opened.

February 5: Emphysema of chest wall and neck noted. Temperature increasing to 102.8, February 7. Part of graft removed. Pus present.

February 26: Remainder of graft extruded. May 2, 1919: Discharged. Civil disability ½. Flail wrist and wearing wristlet at time of discharge.

Failure on account of suppuration. Roentgenograms lost.

Case 2.—A. A., Pvt., Co. C, 3d Army M. P., aged 30. Pistol wound, June 16, 1919, entered Letterman General Hospital, Aug. 18, 1919. Compound comminuted fracture of middle and lower third of right radius. Longitudinal defect. Nonunion.

August 26: Rib graft. Muscular interposition. Three inches of rib tied around fracture. Pleural cavity not opened.

October 31: Cast removed and reapplied.

December 4: Cast removed. Union.

Dec. 27, 1919: Discharged to full duty.

Complete success. Roentgenograms lost.

Case 3.—B. T. P., Pvt., M. G. B., 362d Inf., aged 27, machine gun bullet wound of left forearm, received Sept. 29, 1918; entered Letterman General Hospital, July 3, 1919. Wound healed. Ununited fracture of left ulna in upper third with two-inch defect.

July 10: Rib graft. Six inches of rib split and fragments of ulna fixed in trough. Pleura not opened.

July 18: Sutures removed. Wound looks healthy.

July 26: Pus found in dressings.

October 6: Roentgenogram showing condition of graft (Fig. 2*A*).

November 17: Sequestrums removed.

November 26: Roentgenogram (Fig. 2 *B*).

December 15: Beginning union. Slight discharge.

Jan. 15, 1920: Roentgenogram (Fig. 2*C*).

Jan. 30: Sequestrums removed. Incision packed. Enough callus for union.

February 25: Superficially granulating shallow scar and thick callus noted. Patient has good grip. Good union.

April 14: Since this date patient was reoperated on. Graft refractured during operation.

Partial success. Roentgenograms reveal progressive increase in density of callus.

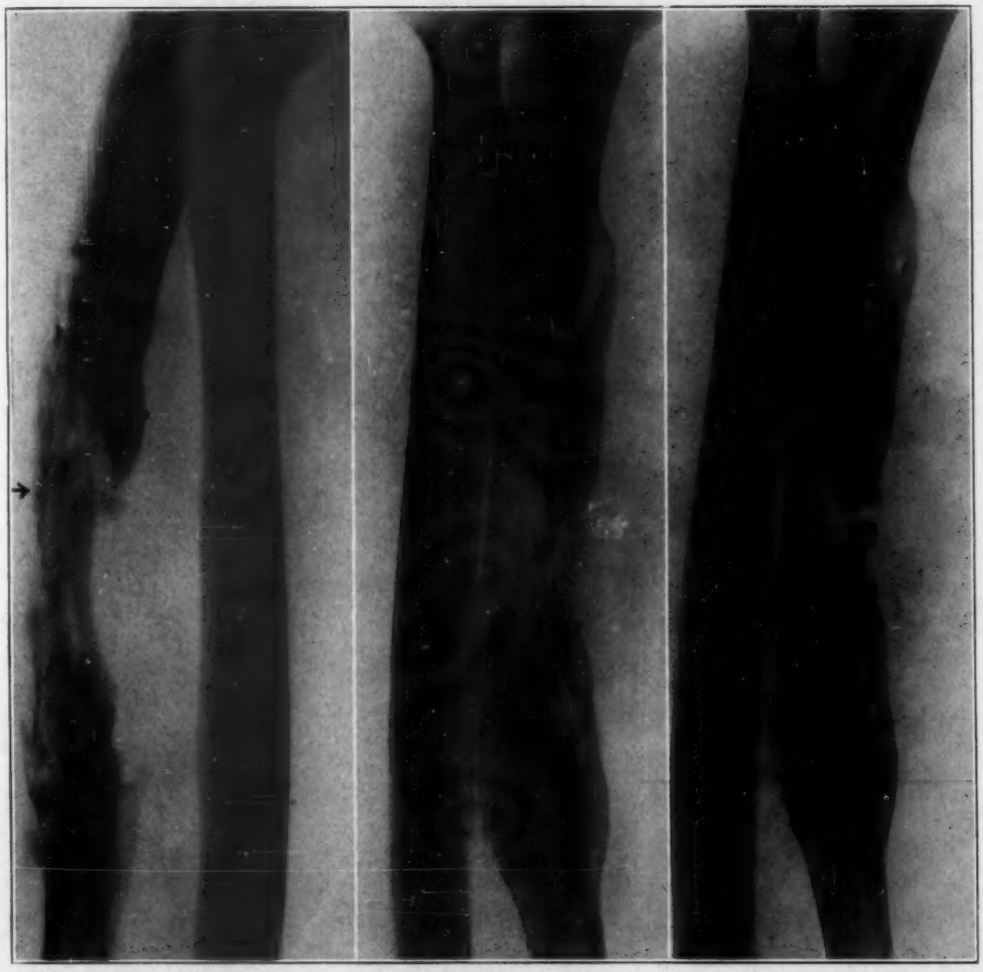
Case 4.—B. P., Cpl., Co. K, 9th Inf., aged 21, high explosive wound received June 29, 1918. Compound comminuted fracture of both bones of left forearm, upper third. Thomas splint applied. Balkan frame used for one and one-half months. Following this, osteotomy was performed for malposition. Nonunion. Infection occurred at a second operation. Entered Letterman General Hospital, Dec. 19, 1918. Flail arm 3 inches below elbow. Nonunion of both bones.

Jan. 21, 1919: Mortising operation. Silver wire passed through drill hole. Primary union of skin incision.

March 1: Nonunion.

May 6: Second mortising operation. Nonunion.

July 25: Roentgenogram: no callus. Separation of fragments of radius. Ulna in fair line, but no callus formation (Fig. 3*A*).



A B C

Fig. 2. (Case 3).—*A*, three months after operation: arrow points to graft with sequestrums. *B*, four months and fourteen days after operation. Firm union and hypertrophy of ends of graft. Osteomyelitis and sequestrum at middle of graft. *C*, six months after operation. Firm union. Refracture occurred later at a reoperation for osteomyelitic sinus.

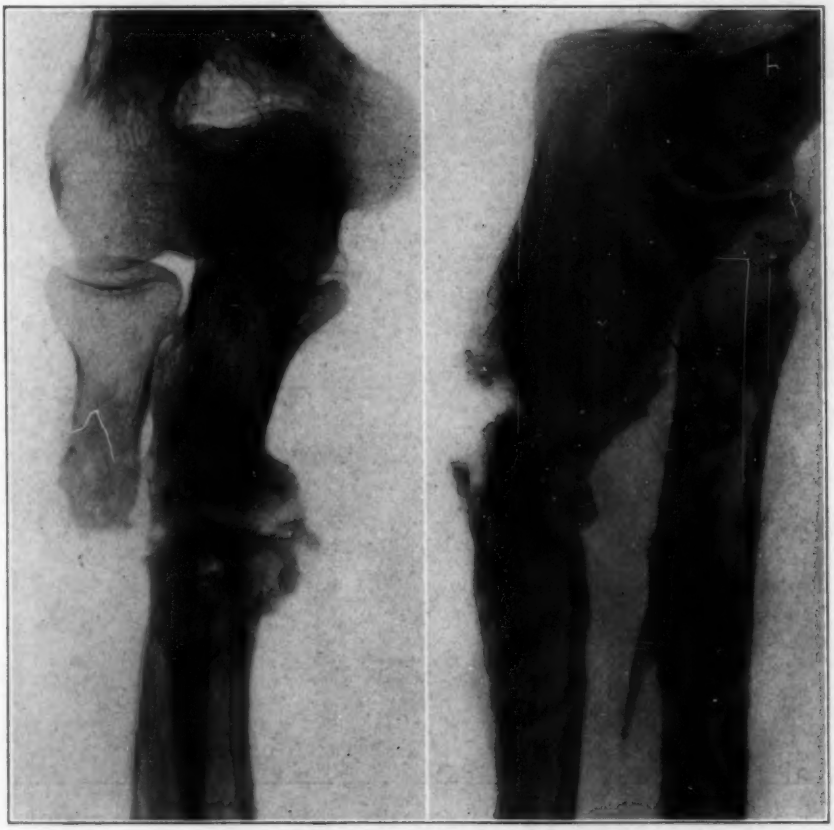
August 5: Third operation. Rib graft inserted. Rib split into halves. Each half chiseled in two diagonally. Four resulting fragments tied around radius and ulna. Pleura opened during removal of ribs.

October 10: Cast removed. Fracture firm. Small pressure sore over graft. Otherwise primary union.

Jan. 8, 1920: Roentgenogram (Fig. 3 *B*).

Jan. 16, 1920: Discharged: (1) shortening of left radius and ulna; (2) incomplete flexion of phalanges. Civil disability: (1) 1/12; (2) 1/24.

April 6: Good strength in arm; almost as good as the other. Full extension. Flexion to 35 degrees. Full pronation. Supination 120 degrees, 60 degrees less than on the normal side. Good grip. Wounds firmly healed. Bones firmly united.



A B

Fig. 3 (Case 4).—*A*, before rib grafting operation. *B*, five months after operation. Firm union. Good alinement.

Complete success.

CASE 5.—C. C., Pfc., Co. I, 361st Inf., aged 24, Sept. 27, 1918, machine gun bullet wound of right humerus, entered Letterman General Hospital, May 15, 1919. Nonunion of the lower end right humerus with a 2 inch defect. Musculospiral palsy is present.

May 24: First operation. Bone freshened and sutured with kangaroo tendon. Musculospiral nerve also sutured.

Nonunion.

August 12: Rib graft inserted. Four and one-half inches of rib split longitudinally and then diagonally. Three of these four pieces were fastened around humerus with kangaroo tendon. Pleura opened during operation. Dec. 28: Roentgenogram (Fig. 4).

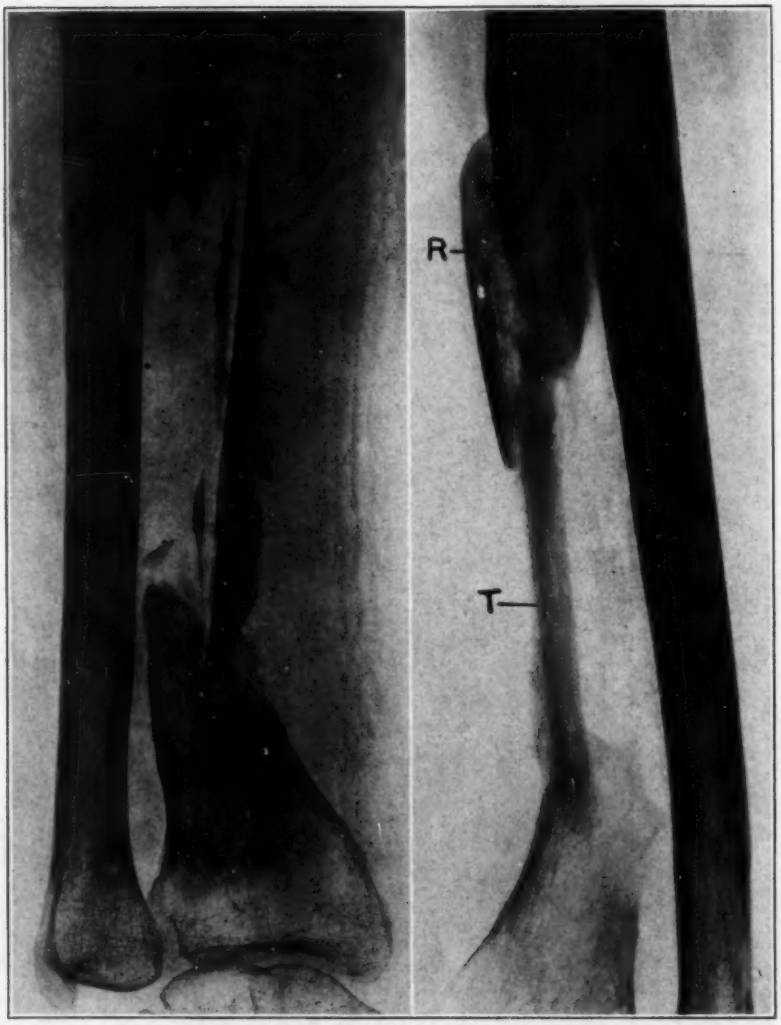
Fig. 4 (Case 5).—Four months and two weeks after operation. Union of grafts at each end; fracture (absorption?) of middle. Nonunion.

October 14: Cast removed. Nonunion.

Jan. 19, 1920: Discharged: (1) musculospiral palsy; (2) humerus is ununited. Civil disability: (1) ¼; (2) ¼.

April 3: Writes that condition is unchanged.

Failure was due to suppuration and absorption of the graft.



A B

Fig. 5 (Case 6).—*A*, twenty-six days after tibial graft. Upper end of graft has slipped. Nonunion. *B*, five months and nineteen days after tibial graft; two months and twenty-two days after rib graft; *R*, rib graft; *T*, tibial graft.

CASE 6.—D. I., Pvt., Co. C, 362d Inf., aged 28, Sept. 29, 1918, high explosive wound of left forearm. Considerable part of radius removed at Evacuation Hospital No. 10.

Jan. 7, 1919: Letterman General Hospital. Eight cm. defect middle and lower third left radius.

Feb. 6: Albee graft from tibia. Distal end wedged into crack. Proximal end fastened to proximal fragment with catgut. Nonunion proximal end.

March 3: Roentgenogram (Fig. 5 *A*).

Fig. 6 (Case 6).—Seven months after tibial graft; four months, fourteen days after rib graft. Firm union of both rib and tibial implants to each other and to matrix bone of radius. Good alinement. *R*, rib graft; *T*, tibial graft.

May 3: Rib graft. Former Albee graft alive. Distal end firmly united. Proximal end lies against radius, separated from it by thin scar. Three inches of sixth rib removed. Rib split. Distal end of proximal fragment of radius and proximal end of graft tied into the rib. Pleura not opened.

July 25: Roentgenogram (Fig. 5 *B*).

Aug. 1: Wound healed; perfectly firm.

Sept. 17: Roentgenogram (Fig. 6).

Sept. 18: Discharged. Ankylosis (partial), left wrist. Contracture flexor muscles fingers of left hand. Civil disability ⅓.

Complete success. Union of second graft to first. Roentgenograms reveal progressive hypertrophy.

Case 7.—D. J. S., Pvt., Co. N, 110th Inf., aged 23, Sept. 4, 1918, received machine gun bullet wound of left forearm, the lower third of the radius being fractured.

Dec. 19, 1918: Entered Letterman General Hospital. Healing scars noted on left forearm. Nonunion left radius with a defect of from 2 to 3 cm.

Feb. 2, 1919: Arm suspended by thumb to correct ulnar deviation of hand.

A B C

Fig. 7 (Case 7).—*A*, twenty-eight days after first operation—chip graft. Before rib grafting operation. *B* and *C*, two months after rib graft operation. Firm union of rib graft. Former chip graft still in place under rib graft. *R*, rib graft; *Ch*, chip graft.

February 6: Operation. Graft 1½ inches long, ½ inch thick, chiseled off outside of radius. One end sharpened into a wedge and forced into a crack made in distal fragment with chisel. Other end laid against upper fragment. No retaining suture. Wound healed by first intention.

March 6: Roentgenogram (Fig. 7 *A*).

May 17: Nonunion. Rib graft. Previous graft separated from each end of radius by scar. Graft is alive. Three and one-half inches of sixth rib removed, split, and tied over fragments of radius. Pleura opened during operation.

July 19: Roentgenogram (Fig. 7 *B* and *C*).

September 4: Firm union. Discharged. Synostosis (partial) between lower end radius and ulna. Ankylosis (partial) metacarpophalangeal joint first finger left. Civil disability ⅛.

Complete success.

A B

Fig. 8 (Case 8).—*A*, three months, nine days after operation. Good alinement. *B*, five months after operation. Firm union. Good callus at end of graft. Good alinement. Increase in density of graft. Compare with A.

CASE 8.—E. R. C., Pvt., Co. C, 59th Inf., aged 28, Oct. 12, 1918, high explosive wound, right forearm. Two sequestrotomies (February and March, 1919).

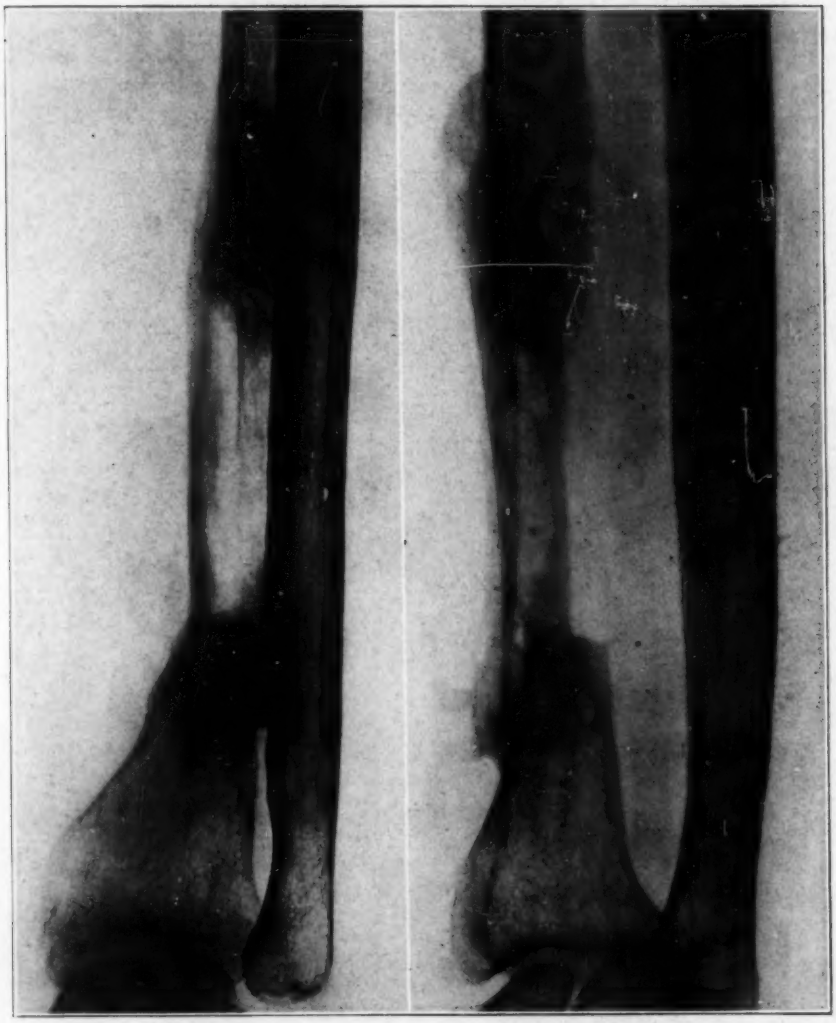


Fig. 9 (Case 8).—Six and one-half months after rib graft. One and one-half months after reoperation at which a piece of graft was removed and the incision packed. Beginning atrophy of graft. Compare with Figure 8. Refracture occurred at a later operation.

June 22, 1919: Entered Letterman General Hospital. Two inch defect, lower fourth of right radius present. Paralysis (partial) right median, ulnar and radial nerves.

July 29: Rib graft. Five inches of rib split and tied about each end of bone.

Aug. 16: Wound suppurating. Wound dressed daily.

Nov. 7: Roentgenogram (Fig. 8 *A*).

Fig. 10 (Case 10).—Two months after operation. Atrophy of graft.

Dec. 15: Firm union.

Dec. 21: Roentgenogram (Fig. 8 *B*).

Dec. 30: Small piece of graft ($\frac{1}{10}$ inch wide and 1 inch long) is loose. This is removed. More of edge of graft removed with chisel. Incision packed and left open.

Feb. 14, 1920: Roentgenogram (Fig. 9).

March 5: Firm union. Small superficial ulcer size of lentil over graft.

March 12: Reoperation. Removal of bone. Refractured at operation. Partial success. Patient in hospital.

CASE 9.—F. L. D., Pvt., Co. K, 308th Inf., aged 29, Oct. 4, 1918, high explosive wound of right shoulder and right jaw.

Feb. 1, 1919: Entered Letterman General Hospital. Defect, right mandible, practically all of vertical ramus and half of horizontal ramus. Large foreign body, upper lobe right lung. Brachial palsy, right.

Fig. 11 (Case 10).—Seven months after operation. Firm union. Graft has had several sequestrums removed from the gaps, shown by the light spots. Increase in density since October, 1919. Compare with Figure 10.

April 1: Rib graft. Posterior end of main fragment of jaw atrophic. Mouth not entered. Four inch piece, curved, of sixth rib removed. Pleural cavity not opened. Anterior end of rib split. Posterior end of main fragment of jaw placed in trough and held with kangaroo suture. Upper end of graft laid into region of condyle of jaw. No fixation of upper end of graft.

April 12: Wound healed.

August 1: Can chew well

August 28: Discharged. Deformity right maxilla; partial ankylosis jaw, unable to chew army rations. Civil disability 1/30.

March 27, 1920: Fibrous band extending backward from site of fracture. It is not possible to feel whether or not bone graft is present in this hard tissue. Jaw gives man no trouble; can crack hard candy with it and can lift chair with his teeth.

Complete success.

CASE 10.—F. A., Pvt., Co. M, 308th Inf., aged 29, Oct. 14, 1918, high explosive wound of left forearm.

Fig. 12 (Case 11).—Thirty days after operation. Good alinement. Union.

July 3, 1919: Admitted to Letterman General Hospital. Sinus leading down to left ulna. Two-inch defect of left radius.

July 12: Sequestrotomy. Wound closed and healed by first intention.

August 7: A rib graft inserted. Four inches of rib removed, split, and tied around radius. Pleura not opened.

August 14: Cast removed. Wound suppurating.

Oct. 7, 1919: Graft united at lower end; ununited at proximal end (Fig. 10).

Jan. 3, 1920: Operation. Unabsorbed part of graft ununited. It is removed and the incision left open.

Feb. 13: Roentgenogram (Fig. 11).

March 15: Graft firmly united. Good function of arm. Wounds healed. Complete success.

CASE 11.—G. H., Pfc., Co. C, 28th Inf., aged 28, July 19, 1918, gunshot wound of left forearm.

Sept. 1, 1918: Healed, but reopened afterward for recurring suppuration.

Feb. 20, 1919: Admitted to Letterman General Hospital, where compound comminuted fracture of the middle third left radius with a small defect was found. Partial interruption of median and ulnar nerve also noted.

Fig. 13 (Case 13).—Before rib grafting operation; after implantation of bone chips.

A sequestrotomy was first performed, then between Aug. 2 and 15, 1919, a rib graft was inserted by Captain Haas.

September 15: Roentgenogram (Fig. 12).

Nov. 18: Discharged. Partial paralysis of median and ulnar nerves. Partial ankylosis of left wrist and of all carpal and phalangeal joints. Weakness of left hand. Civil disability ⅓. Bone graft firmly united. Records lost. Complete success for bone graft.

CASE 12.—H. E. S., First Lieut., Co. F, 58th Inf., aged 28, Oct. 5, 1918, high explosive wound of right forearm. Bone graft from tibia inserted by Major Albee.

March 24, 1919: Primary union. Radius refractured.

June 23: Letterman General Hospital. Ununited fracture of radius and ulna, lower third.

July 8: Operation performed by Captain Haas. Tibial graft united at lower end. Nonunion, upper end. Graft in upper fragment being absorbed. Easily pulled out of bed, but surrounded by new bone. Piece of rib excised, wedge cut in upper fragment, end of rib anchored into same. Lower end split, lower fragment inserted into trough.

Fig. 14. (Case 13).—Refracture of graft at original site of nonunion. Part of graft distal to refracture was absorbed by March 13, 1920. Compare with Figure 15.

July 23: Serous discharge.

September 23: Sinuses. Sequestrums removed. Both ends of graft ununited.

October 18: Third operation. Remains of graft united to surrounding tissue. Ridge of new bone under graft continuous with lower fragment, in contact with upper fragment, but not united.

Still in hospital. Nonunion. Failure because of suppuration.

Case 13.—J. O. M., Pvt., Co. K, 308th Inf., aged 29, Sept. 29, 1918, high explosive wound of left forearm.

September 30: Bone fragments removed.

Dec. 18, 1918: Admitted to Letterman General Hospital. Nonunion of radius.

Jan. 20, 1919: Operation. Small spike chiseled out from proximal fragment and jammed into defect. Remaining space filled with bone chips.

Nonunion.

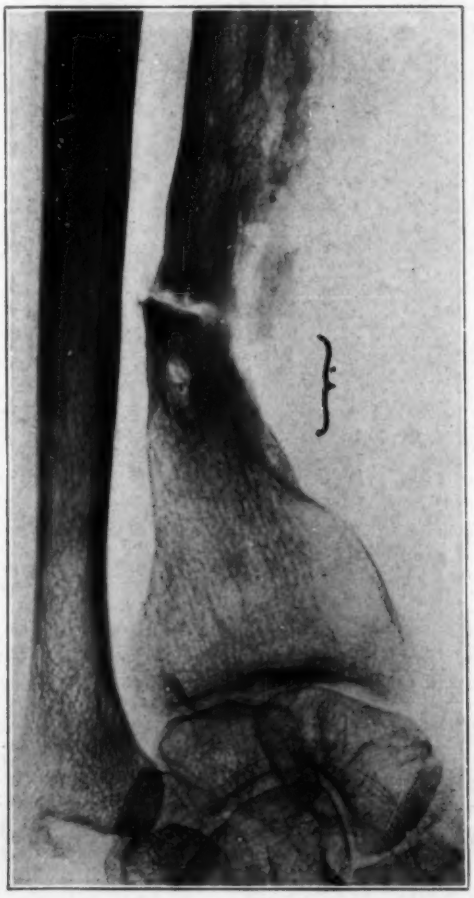


Fig. 15 (Case 13).—Firm union, upper end of graft. Refracture through lower end at site of original nonunion. Absorption of graft distal to site of refracture. Compare with Figure 14. Bracket indicates part of graft that has been absorbed.

July 31: Second operation. Rib graft. Fibrous separation ⅛ inch; ¾ inch of bone removed. Piece of rib 2½ inches removed, split, and fragments tied into trough. Pleura not opened.

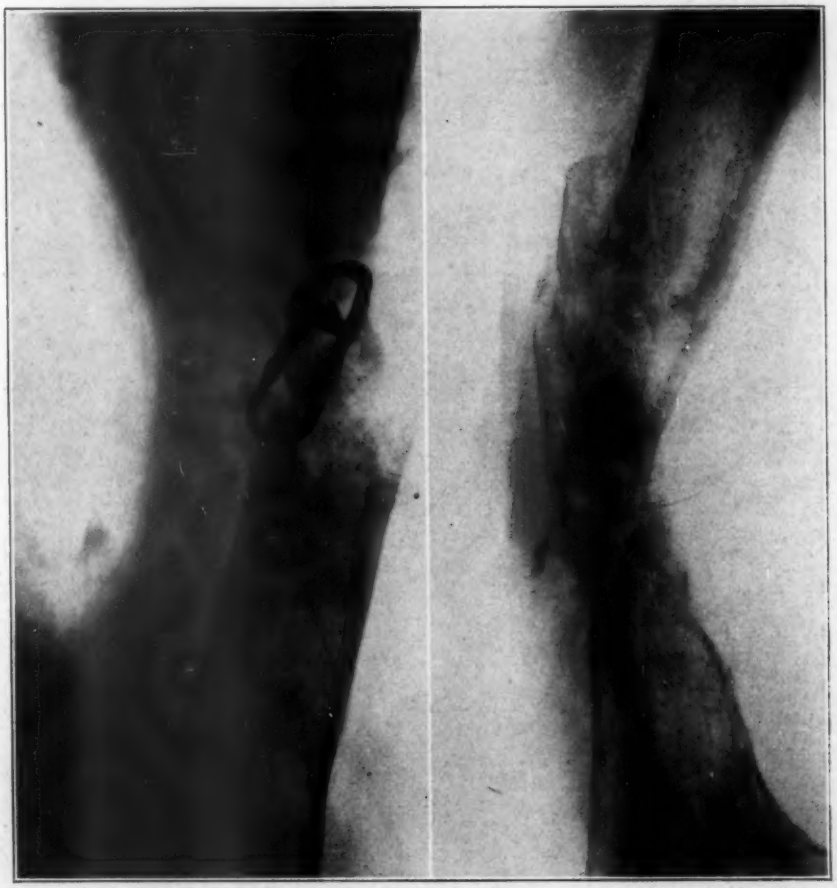
Jan. 9, 1920: Discharged. Defect of radius, junction middle and lower third. Destruction of flexor and extensor muscles. Ankylosis (partial) wrist. Civil disability ⅓.

March 13: Graft firm at both ends. Fracture in middle. Good grip.

Partial success. Fracture of graft.

CASE 14.—J. A., Pvt., Co. I, 111th Inf., aged 31, Oct. 2, 1918, machine gun bullet wound of left arm.

October 3: Bone removed at evacuation hospital. Musculospiral palsy.



A B

Fig. 16 (Case 14).—*A*, before grafting operation. *B*, eight weeks after operation. Sepsis. Absorption and disintegration of graft.

Jan. 17, 1919: Letterman General Hospital. Nonunion of humerus. Enormous defect of middle third. Partial musculospiral palsy.

March 18: Neurolysis radial nerve. Bone suture with silver wire.

June 12: Nonunion.

August 19: Rib graft. Four pieces of rib fastened about fragments. Pleura not opened. Infection and copious drainage.

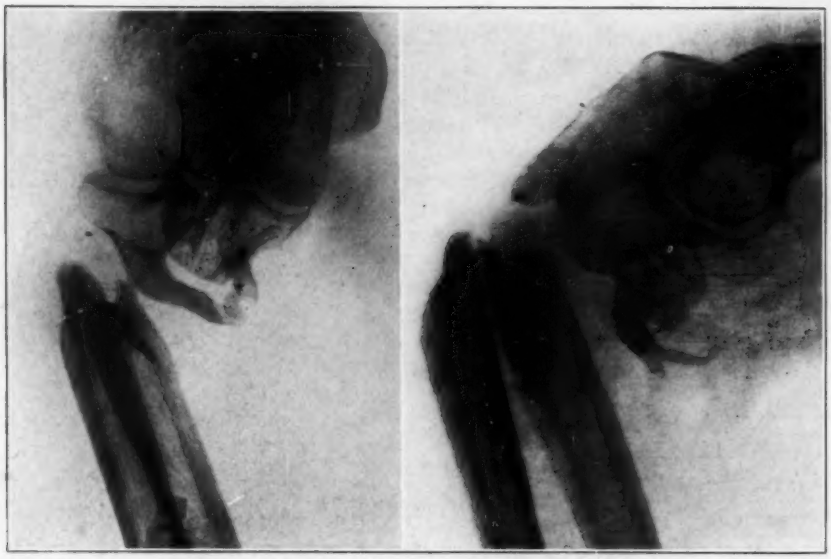
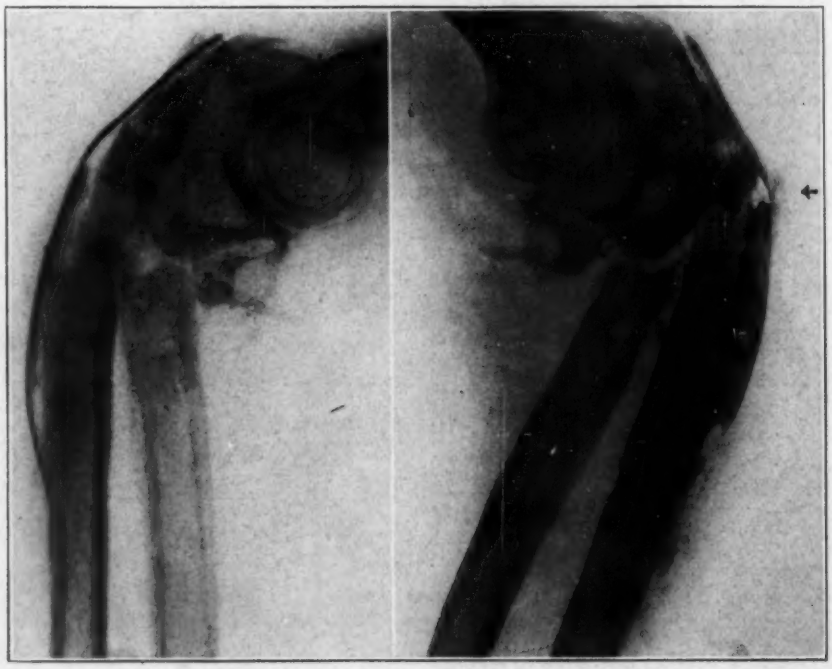


Fig. 17 (Case 15).—Before operation.



A B

Fig. 18 (Case 15).—*A*, eleven weeks after operation. Union. Good alinement. *B*, fracture and absorption of graft through middle; arrow points to new callus about site of fracture of graft. Firm union, both ends. Six months after operation.

August 21: Sequestrotomy.

October 15: Roentgenogram (Fig. 16 *B*).

Dec. 10, 1919: Nonunion.

March 5, 1920: Flail arm. Remains of graft in good position and united to upper and lower fragments by callus. Fragments run out into sharp spikes. Good grip. Radial palsy cured.

Failure.

CASE 15.—L. H. B., Pvt, Co. E, 309th Inf., aged 25, Nov. 1, 1919, machine gun bullet, left elbow. Débridement and wound healed, February, 1919.

Fig. 19 (Case 16).—Twenty-eight days after Albee operation. Lower end of tibial graft slipped from slot into which it was implanted.

July 3, 1919: Letterman General Hospital. Flail elbow, left. Wrist drop, left. Large defect.

July 19: Rib graft. One and one-half inch defect of ulna bridged over by 4½ inches of sixth rib. Head of radius inaccessible. Small sliver of rib laid against it, but does not bridge gap accurately. Pleura opened.

Sept. 12: Beginning union. Wounds healed.

October 7: Roentgenogram (Fig. 18 *A*).

Oct. 10: Firm union.

Jan. 20, 1920: Roentgenogram (Fig. 18 *B*).

Results in Twenty-Two Cases of Rib Graft

Case No.	Name	Kind of Injury; Region; Date*	Size of Defect	Operation			Time Between Injury and Graft	Time Between Wound Healed and Graft	Suppuration	Time Between Graft and Union	Time in Cast	Refracture	Result	Remarks	Surgeon's Certificate of Disability
				First	Second	Third									
1	A. O.	H. E., wrist, 5/4/18	All carpals except trapezium	Rib graft, 2/3/19	Graft removed, 2/26/19	.....	8 mos.	.....	Yes	.....	....	.....	Failure, acct. suppuration	Subcutaneous emphysema	Leather wristlet, flail wrist, C. ½† 5/2/19
2	A. A.	G. S. W., middle and lower 3d radius, 6/16/19	Small	Rib graft, 8/18/19	.....	.....	2 mos.	.....	No	3 mos.	3 mos.	No	Success	.....	To full duty 12/22/19
3	B. T. P.	G. S. W., middle ulna, 9/29/18	2 in.	Rib graft 7/10/19	Sequestrotomy, 11/7/19	Sequestrotomy, 1/30/20	9½ mos.	.....	Yes	3 mos.	....	At reoperation Mar. '20	Partial success	Reoperated later with removal of bone	Still in hospital, 3/28/20; operated and refractured (indication for operation ?)
4	B. P.	H. E., ulna and radius, upper third 6/29/18	.....	Freshened and wired, 1/21/19	Mortise, 5/6/19	Rib graft, 8/5/19	13½ mos.	About 11 mos.	No	2 mos.	2 mos.	No	Success	Two previous osteotomies for malposition; second one infected	C. 1/12, 1/16/20
5	C. C.	M. G. B., lower third humerus, 9/29/18	2 in.	Bone suture, 5/24/19	Rib graft, 8/12/19	.....	10½ mos.	.....	....	.....	....	Yes	Failure	Musculospiral palsy	S. C. D., 1/19/20: (1) palsy musculospiral, C. ¼; (2) nonunion humerus, C. ¼
6	D. I.	H. E., middle and lower third radius, 9/29/18	3½ in.	Albee, 2/6/19	Rib graft, 5/13/19	.....	(1) 4½ mos. (2) 7 mos.	.....	No	3 mos.	....	No	Success	.....	C. ⅓, 9/18/19, ankyl. wrist, tendon contracture
7	D. J. S.	H. E., middle and lower third radius, 9/4/18	1 in.	Chip graft 2/6/19	Rib graft, 5/17/19	.....	8½ mos.	(1) 1 mo. (2) 4½ mos.	No	3 mos.	....	No	Success	.....	C. ⅛, 9/14/19, synostosis (partial) radius and ulna; fibrous ankylosis (partial) second metacarpophalangeal joint
8	E. R. C.	H. E., middle and lower third radius 10/12/18	1¾ in.	Rib graft. 7/29/19	Sequestrotomy, 12/30/19	Bone removed 3/12/20	9½ mos.	.....	Yes	4½ mos.	....	Yes (at oper.)	Partial success	Reoperation after firm union (reason ?) refractured at operation	Ulnar and median palsy, still in hospital
9	F. L. D.	H. E., jaw 10/14/18	½ of jaw	Rib graft, 4/1/19	.....	.....	7 mos.	3 mos.	Yes	.....	....	.....	Success	Large foreign body of chest; brachial palsy (partial)	S. C. D., C. 1/30, 8/28/19; 5 mos. post. oper.
10	F. A.	H. E., middle third radius 10/14/18	1 in.	Sequestrotomy, 7/12/19	Rib graft, 8/7/19	Sequestrotomy, 1/3/20	11 mos.	1 mo.	Yes	(1) 3 mos. (2) 7 mos.	....	No	Success	Suture after sequestrotomy	Still in hospital; wounds healed

11	G. H.	G. S. W., middle third radius, 7/19/18	½ in.	Rib graft between 5/2-15/19	.....	.....	12½ mos.	.....	No	3 mos.	.... *	No	Success	Ulnar and median palsy (partial)	S. C. D., palsy (partial) rad. and ulnar nerves; ankyl. (partial) all carpal and phalangeal joints; C. ⅛, 11/15/19
12	H. E. S.	H. E., lower third radius, and ulna, 10/5/18	.....	Rib graft (Capt. Haas), 7/8/19	Incision, 9/23/19 graft alive	Incision, 10/18/19; graft alive	9 mos.	.....	Yes	Nonunion upper end	2 wks.?	.....	Failure	Bone graft (Major Albee), 3/24/15; rib jammed into wedge, upper end	Still in hospital
13	J. O. M.	H. E., lower third radius, 10/29/18	..... 1/28/19	Chip graft	Rib graft 8/31/19	.....	10 mos.	.....	Late	.....	....	Yes	Partial success	Destruction muscles	S. C. D., 1/9/20; defect left rad.; destruction rad. reflex and exten. muscles; C. ⅛
14	J. A.	M. G. B., lower and middle third humerus, 10/2/18	⅜ in.	Wired, 3/18/19	Rib graft, 8/19/19	Sequestrotomy, 10/21/19	10½ mos	.....	Yes	Nonunion	....	.....	Failure	Radial palsy (partial)	Still in hospital
15	L. H. B.	F. G. B., radius and ulna, upper third, 11/11/18	1 in.	Rib graft, 8/19/19	.....	.....	8½ mos.	3 mos.	No	2 mos.	....	Yes	Success	Radial palsy	Still in hospital (wrist drop)
16	M. K.	M. G. B., middle third radius. 9/29/18	1¾ in.	Albee, 3/6/19	Rib graft, 5/15/19	.....	7½ mos.	.....	No	2¾ mos.	....	No	Success	Partial inteross. and median palsy; Albee too small, slipped in cast	S. C. D., C. ⅛, partial median palsy, 9/26/19
17	O. W. J.	H. E., lower third radius, 9/29/18	2 in.	Sequestrotomy; sterile pus. 8/24/19	Rib graft, 8/28/19	.....	11 mos.	6 mos.	No	6 wks.	....	No	Success	Ulnar palsy and suture, 5/15/19	Still in hospital
18	R. T.	H. E., middle of tibia, 11/1/18	2 in.	Rib graft, 8/31/19	.....	.....	9 mos.	2 wks. (barely healed)	Yes	2¼ mos., upper and lower end	....	No	Success	Temperature reaction; no sequestrums	Still in hospital; drop foot; loss ext. tendons
19	R. L.	H. E., middle third radius, 8/30/18	2 in.	Rib graft, 10/18/19	. .....	... .	14½ mos.	.....	Late	.....	....	Yes	Partial success	Pus on 7th day; sinus closed 6 wks. later; no sequestrums	Still in hosp.; refracture; post. inteross. palsy; S.C.D., 3/13/20, C. ⅛, ankylosis (partial) wrist
20	S. T.	C. S. W., upper third humerus, 10/28/19	2½ in.	Rib graft, 8/12/19	.....	.....	9½ mos.	4 mos.	Late	.....	....	Yes	Partial success	Pus 1 mo. after oper.; hemolyt. strept.	Still in hospital; fracture; fibrous union; sinus closed, Jan. '20, following erysipelas
21	S. W.	C. C. F., ulna and radius, lower third, 8/15/18	Slight	Chip graft, 6/12/19,	Rib graft, 8/15/19	.....	17 mos.	4 mos.	No	1 mo. beginning, 2 mos. firm	....	No	Success	Two previous operations in other hospitals	S. C. D., C. 1/10, ankylosis (partial) wrist, 11/14/19
22	S. E.	C. C. F., ulna and radius, upper third, 10/4/18	Slight	Rib graft, 4/19/19	.....	.....	6½ mos.	.....	No	3 mos.	....	No	Success	Nonunion lower end of graft, but good function	Discharged 8/14/19

* In this column, H. E. represents high explosive; G. S. W., gunshot wound; M. G. B., machine gun bullet, and C. C. F., compound comminuted fracture.
† C.½ represents civil disability, one half.

Jan. 28: Graft fractured at middle.

Feb. 17: Broken graft uniting.

Feb. 25: Firm union of ulna. Nonunion of radius. Good power in elbow. Wrist drop. Still in hospital for wrist drop.

Success.

Case 16.—M. K., Pvt., Co. H, 147th Inf., aged 31, Sept. 29, 1918, machine gun bullet, left forearm. Partial paralysis of posterior interosseus.

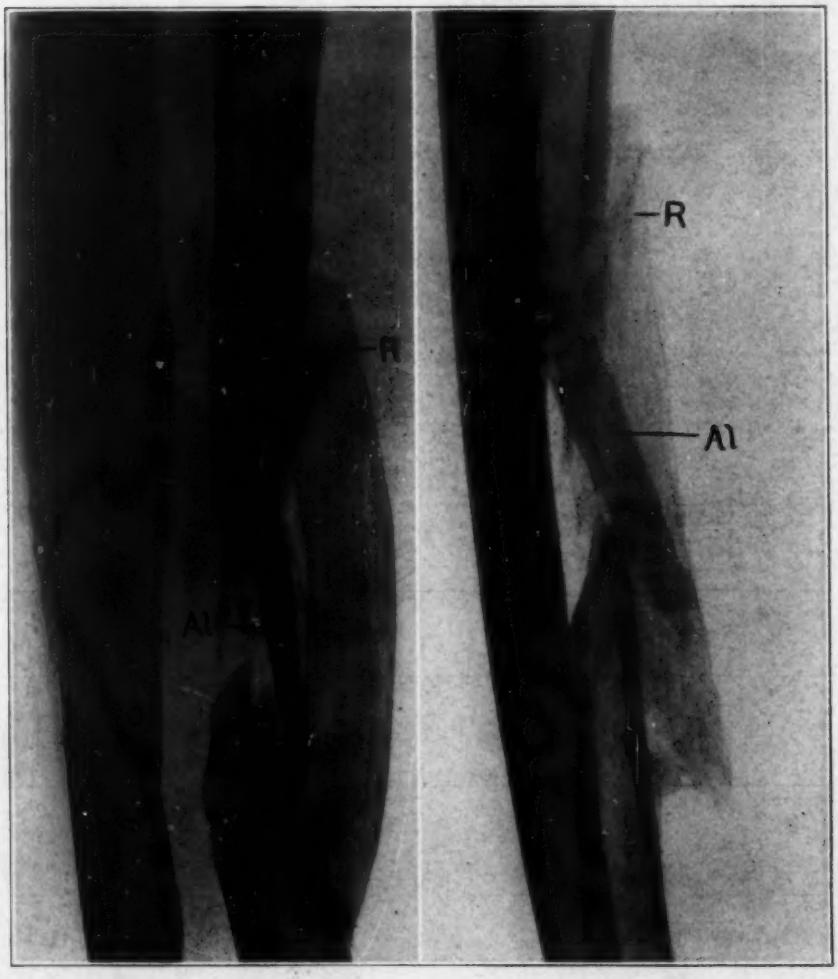


Fig. 20 (Case 16).—Firm union of rib graft. Previous Albee graft lies under rib graft. Three months after rib graft operation. *R*, rib graft; *Al*, Albee graft.

Jan. 7, 1919: Admitted to Letterman General Hospital. Three cm. defect middle third left radius.

March 6 Albee graft removed from tibia; edges sharpened into wedge, forced into crack in each fragment. No suture.

April 3: Roentgenogram (Fig. 19).

April 26: Upper end of graft became loose. Soon after operation displaced from crack.

May 15: Rib graft. Previous graft ununited at both ends. Distal end firmly embedded in slot made at operation March 6. Four inches of sixth rib removed, split and tied about bone. Original tibial graft left in place. Minute opening in pleura.

August 8: Firm union. Discharged. Partial paralysis of median nerve. Civil disability ⅛.

August 15: Roentgenogram (Fig. 20).

Complete success.

A B C

Fig. 21 (Case 17).—*A*, before operation. Radial deviation of wrist. *B*, forty days after operation. Correction of radial deviation. *C*, five and one-half months after operation. Firm union, ends of graft. *F*, fracture through middle of one graft.

CASE 17.—O. W. J., Sgt., Co. L, 363d Inf., aged 25, Sept. 29, 1918, two inches of radius carried away by shell. Fragments removed same day.

April 12, 1919: Letterman General Hospital. Two-inch defect lower end right radius. Partial ulnar palsy, right.

May 15: Resection and suture of ulnar nerve.

July 24: Sequestrotomy of radius.

August 28: Rib graft. Five inches of seventh rib split and fastened around radius. Pleura not opened.

Sept. 18: Primary union.

Oct. 7: Union firm (Fig. 21 *B*).

Feb. 4, 1920: Roentgenogram (Fig. 21 *C*).

March 5: Firm union. Some projection ulnar styloid and volar displacement of carpals. Dorsal extension of wrist impossible beyond straight line. Partial ulnar palsy.

Complete success.

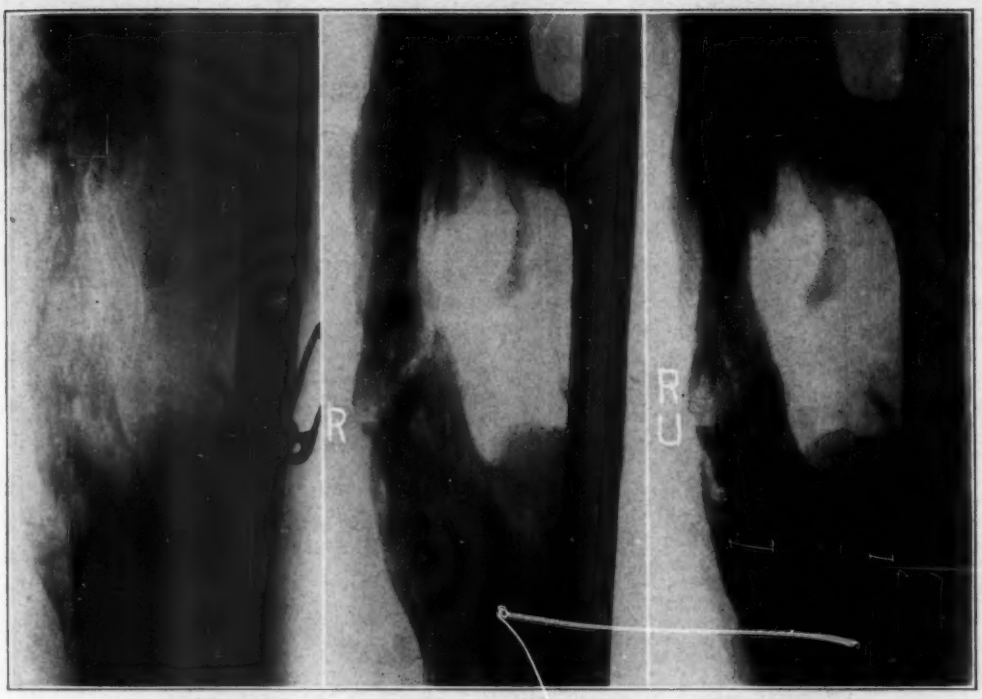
Fig. 22 (Case 18).—Tibial defect before operation. Synostosis of upper tibial fragment to fibula.

CASE 18.—R. T., Pfc., Co. E, 361st Inf., aged 24, Nov. 1, 1918, high explosive wound of right tibia.

July 4., 1919: Letterman General Hospital. Wound over middle right tibia. Small granulating area. Defect 2 inches long in middle of right tibia.

July 31: Rib graft. Incision on inside of leg behind old scar. Back of tibia exposed. Part of scar separating ends of bone excised. Five inches of rib removed, split, and laid against back of tibia. Kangaroo tendon encircling rib passed through drill hole in tibia. Long posterior splint. Pleura not opened.

August 8: Wound suppurating.



A B C

Fig. 23 (Case 18).—*A*, nine weeks after rib graft. *B*, five and one-half months after rib graft. Fracture of graft through its middle, in spite of much increase in density. *C*, six and one-half months after operation. Beginning union of refracture of graft. *R*, refracture; *R U*, refracture united.

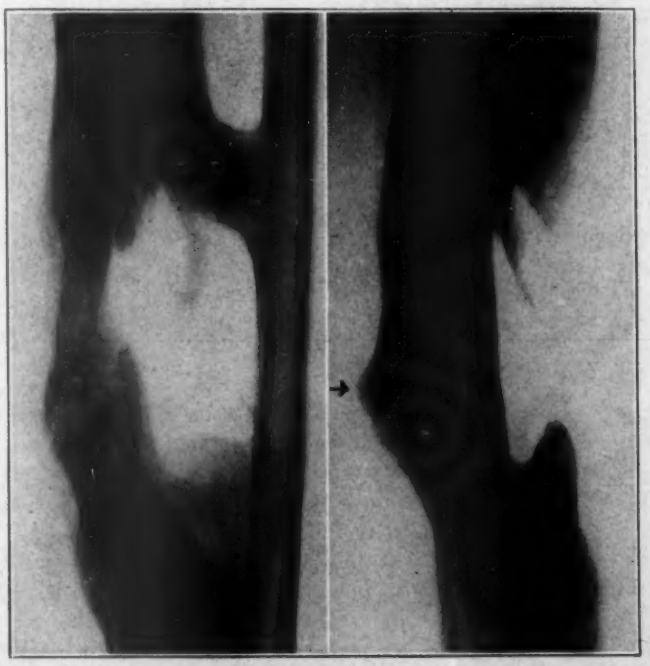
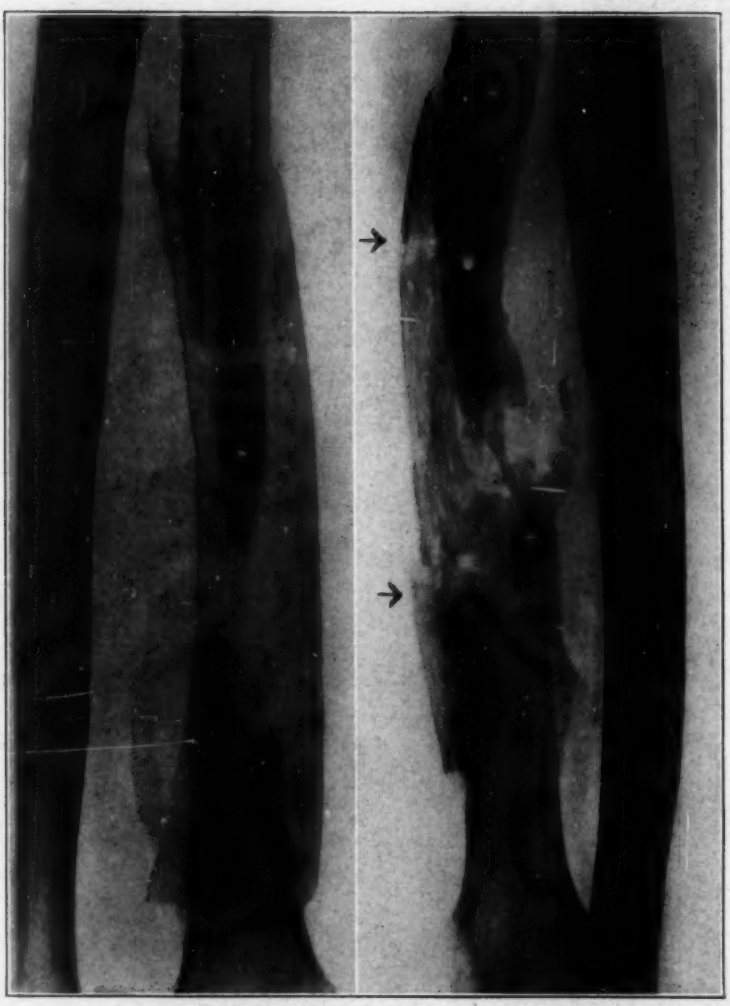


Fig. 24 (Case 18).—Nine months after operation. Union of refracture of graft with considerable callus. Arrow points to callus at site of fracture of graft.



A B

Fig. 25 (Case 19).—*A*, thirty-seven days after operation. *B*, three and one-half months after operation. Arrows point to absorption of graft at site where kangaroo tendon was tied. Refracture occurred later at these places. Pressure atrophy (?). Suppuration about kangaroo tendon (?).

August 9: Chill.

August 12: Temperature normal. Wound gradually closed without sequestration.

October 6: Roentgenogram (Fig. 23 *A*).

Oct. 8: Firm union upper end of graft. Lower end movable. Wound closed. Double bar brace, gradual weight-bearing.

Jan. 13, 1920: Fracture of graft (Fig. 23 *B*).

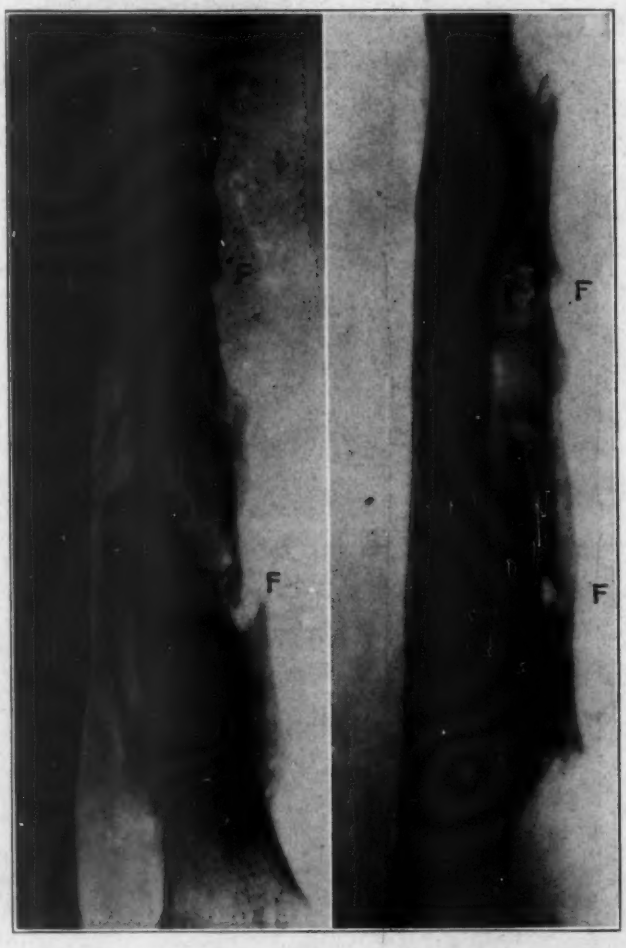


Fig. 26 (Case 19).—Firm union, both ends of graft. *F*, fractures at the middle.

February 17: Beginning reunion.

March 5, 1920: Walks with brace, but without stick or crutch. Ridge of bone both on inside and outside of tibia. Synostosis with fibula as a bridge of bone separate from graft. Sinus closed. Drop foot from loss of muscle.

Complete success. Patient can stand on grafted leg alone.

CASE 19.—R. L., Pvt., Co. L, 109th Inf., aged 27, July 30, 1918, high explosive wound of left forearm.

Aug. 10, 1919: Letterman General Hospital. Wounds healed. Compound comminuted fracture of radius. Partial median paralysis.

Fig. 27 (Case 20).—Large defect, upper third of humerus. Before operation.

October 18: Rib graft. Small defect. Large soft callus. Four inches of rib split and tied around defect. Tendon transplantation at same operation. Pleura not opened.

October 31: Primary union.

November 4: Pus.

A B

Fig. 28 (Case 20).—*A*, union, upper and lower portions of graft. Beginning absorption of middle. Four months after operation. *B*, union of upper and lower portions of graft. Refracture through middle. Six months after operation.

November 29: Roentgenogram (Fig. 25).

Dec. 20: Closed.

Refracture of graft some time after February 20, 1920. Fracture of graft in middle. Both ends firmly welded to radius (Fig. 26).

March 13, 1920: Discharged. Partial ankylosis of wrist. Civil disability 1/8.

March 29: Nonunion, but strong arm.

Partial success.

CASE 20:—S. T., Pvt., Co. D, 58th Inf., aged 25, Sept. 28, 1918, gunshot wound of upper and middle third of left humerus. Bone removed next day.

July 4, 1919: Letterman General Hospital. Two inch defect below head of humerus. Accompanied by much soft tissue destruction. No nerve lesion.

A B

Fig. 29 (Case 21).—*A*, before operation. Nonunion. *B*, rib graft, twenty-nine days after operation.

July 12: Rib graft. Ends of humerus freed of periosteum. Four inches of fifth and sixth ribs, left, removed. Ribs split in half, each half fastened to ends of humerus by two kangaroo tendon sutures. Space between grafts (new marrow cavity) filled with blood clot and iodoform powder. Pleura not opened.

August 15: Slight discharge.

November 4: Roentgenogram (Fig. 28 *A*).

Jan. 9, 1920: Small sequestrums removed.

January 12: Roentgenogram (Fig. 28 *B*).

March 5: Sinus healed after an attack of erysipelas in January. Head rotates with shaft, but lateral mobility present. Nonunion. Spike extends upward from lower fragment. Refracture or absorption of graft.

Failure.

CASE 21.—S. W. A., Pvt., 64th Balloon Company, March 15, 1918, fracture of radius and ulna, lower third, following balloon accident. Two operations for nonunion.

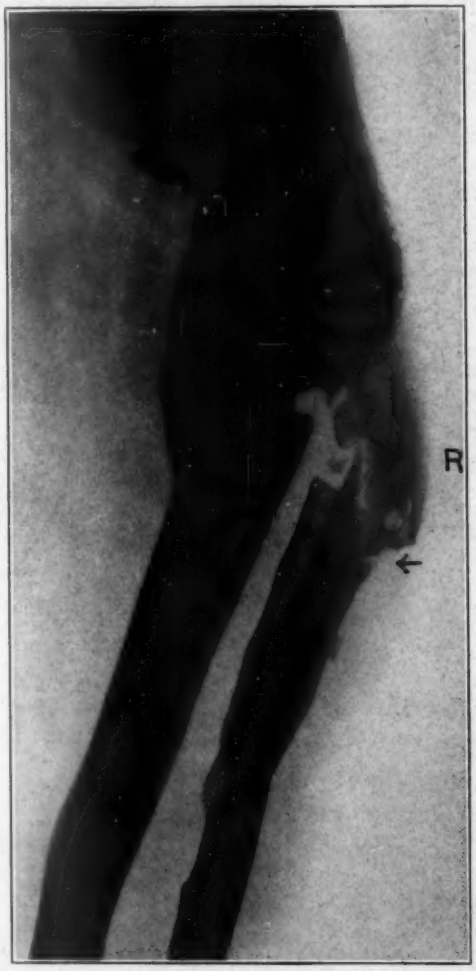


Fig. 30 (Case 33).—Rib graft, four months after operation. *R*, rib graft. Arrow points to beginning union.

Feb. 13, 1919: Letterman General Hospital. Ununited fracture of radius and ulna, lower third. Small defect. Open scar. Silver wire in sinus.

June 12: First operation. Fracture exposed. Small sequestrums removed. Bone chips laid between fragments. Nonunion.

August 5: Rib graft. Three inches of rib split and tied about radius. Pleura opened, small hole.

September 3: Roentgenogram (Fig. 29 *B*).

September 5: Beginning union.

October 7: Union firm. Primary union of skin.

November 4: Discharged. Ankylosis (partial) right wrist. Pain and weakness. Civil disability 1/10.

Complete success.

CASE 22.—S. E., Ensign, U. S. N. R. F., aged 22, Oct. 4, 1918, compound comminuted fracture of ulna and radius, right, upper third, aeroplane propeller.

October 29: Fragment removed.

December 26: Letterman General Hospital. Partial ankylosis of elbow. Wide separation of ulnar fragments.

April 19, 1919: Rib graft. One inch defect. Two inches of sixth rib, left, removed. Slot ½ inch long in upper fragment for reception of graft. Graft jammed into slot with hammer. Lower end of rib split and tied around distal fragment. Primary union.

March 31, 1920: Firm union. Extension to 170 degrees; flexion to 45 degrees; 20 degrees rotation. Success. Can play golf; normal use of arm.

135 Stockton Street.

THE ESTABLISHMENT OF SURGICAL PRINCIPLES IN THE TREATMENT OF FRACTURE OF THE NECK OF THE FEMUR

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NEW YORK

In reviewing the general literature on the subject of fractures and their treatment, one is impressed with the fact that one injury, and only one, is regarded as a hopeless proposition—fracture of the neck of the femur. The more modern authors mention various forms of treatment and concede the possibility of the union of such a fracture, but the general tone adopted is one of discouragement and disillusionment. In one of our best known textbooks,[1] the section on treatment of this injury begins: "The first indication is to save life." In the section on fractures of the skull and of the pelvis, notably grave conditions, the author says nothing about life saving as a primary indication for treatment. Indeed, one would suppose that life saving was the essential preliminary to any treatment, and that it might be taken for granted. Why, then, is its necessity emphasized in connection with this one injury?

In answering this question, one must review the history of the injury. I take as my premise that fracture of the neck of the femur is now very generally regarded as: (1) an injury of advanced age; (2) an injury for which there is no standard treatment, and, (3) an injury differing from others in that the impression is widely prevalent that an intracapsular fracture of the neck of the femur is per se incapable of repair.

It is extraordinary that one bone in the skeleton should be distinguished for its lack of a characteristic accorded to all others. As an explanation of this invidious distinction, I can do no better than to quote at length from the author whose opinion, wrongly, I believe, has always been adduced in support of the proposition that intracapsular fracture of the neck of the femur could not unite.[2] Sir Astley Cooper[2] said:

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1. Stimson, L. J.: Fractures and Dislocations, Ed. 6, Philadelphia, Lea & Febiger, 1910, p. 354.

2. Cooper, Astley: Dislocations and Fractures, Ed. 1, London, John Churchill, 1822.

Much difference of opinion has existed on the subject of the union of the fractured neck of the thigh bone. It has been asserted that these fractures unite like those of other parts of the body; but the dissections which I made in early life and the opportunities I have since had of confirming these observations, have convinced me that fracture of the neck of the thigh bone, of the patella, olecranon, condyle of the os humeri, and of the coronoid process of the ulna generally unite by ligament and not by bone. This principle I taught in my lectures for thirty years: and it is a most essential point, as it affects the reputation of the surgeon, as well as being a subject of some interest in forensic medicine; for if these fractures unite like those of other parts of the body, the patient who remains lame after the treatment would undoubtedly have a right to such redress from the surgeon at the hands of the law.

Nothing is known in our profession by guess; . . . it is right, therefore, that those who are studying their profession should be aware that there is no short road to knowledge: that observation upon the diseased living, examinations of the dead, and experiments upon living animals are the only sources of true surgical knowledge and that indications from these are the sole bases of legitimate theory.

In the examinations which I have made of transverse fractures of the cervix femoris entirely within the capsular ligament, I have only met with one in which a bony union had taken place or which did not admit of a motion of one bone upon the other. To deny the possibility of this union, and to maintain that no exception to the general rule can take place, would be presumptuous . . .

Much trouble has been taken to impress the minds of the public with the false idea that I have denied the possibility of union of the fracture of the neck of the thigh bone: and I therefore beg at once to be understood to contend for the principle only, that I believe the reason that fractures of the neck of the thigh bone do not unite is, *that the ligamentous sheath and periosteum of the thigh bone are torn through,* that the bones are consequently drawn asunder by the muscles, and that there is a want of nourishment of the head of the bone, but I can readily believe that if a fracture should happen without the reflected ligament being torn, that as the nutrition would continue, the bones might unite: but the characters of the accident would differ; the nature of the injury could scarcely be discerned, and the patient's bone would unite with little attention on the part of the surgeon.

Cause of the want of union, first, is the want of proper apposition of the bones, for if in any part of the body the extremities of a broken bone be kept asunder ossific union will under ordinary circumstances be prevented . . .

The neck of the thigh bone, when broken, is placed under similar circumstances (to the fractured patella) for, by contraction of the muscles it is no longer in apposition with the head of the bone and is therefore prevented from uniting; if this, however, were the only obstacle, it might be argued that the retraction of the thigh bone could be prevented by bandaging and extension, the truth of which cannot be denied; but it is scarcely possible, even for a few hours, to preserve the limb in exact apposition, as the patient, on the slightest change of posture, produces instant retraction by bringing into action those powerful muscles, which pass from the pelvis to the thigh bone . . .

A second circumstance which prevents the bony union of these fractures is want of pressure of one extremity of the broken neck upon the other, even though the limb preserves its length, and the fractured parts are consequently

not much displaced. This want of pressure may arise in some degree from the excessive secretion of serous synovia which follows the accident; but principally from the action of the muscles which separate the bony surfaces.

A third reason which may be assigned for the general want of union of this fracture is the almost entire absence of nutrition in the head of the thigh bone when separated from its cervix, its life being supported by the ligamentum teres, which has only a few minute vessels ramifying from it to the head of the bone.

The fourth reason is . . . want of constitutional vigor which always accompanies old age.

It seems strange that a Fellow of the Royal Society, who could so clearly summarize the results of his observations, did not accomplish the final step in his reasoning—that all the obstacles enumerated against repair were due simply to lack of command of an effective treatment. But in Sir Astley's day the neck of the femur was not the only bone "*generally uniting by ligament.*" It shared the burden with the "patella, the olecranon, the condyles of the os humeri, and the coronoid process of the ulna." One by one, as the science of treatment, particularly in the sphere of open operation, progressed, these other bones have been shown, when their fractured surfaces have been placed and held in apposition, to be capable of bony union.

In 1904, it was demonstrated that under favorable circumstances the neck of the femur might also so unite. In other words, a proper form of treatment was for the first time described.[3] Originally devised for the treatment of fractures in children, or in young and vigorous adults, its application has been gradually expanded until now it has been successfully applied to all classes of cases.

That this treatment has not been widely adopted is a source of surprise and regret to those familiar with it. The present status of treatment of fractures of the neck of the femur is evidenced by the subjoined quotation:[4]

After carefully reviewing the case records of these patients, the outstanding points established were that the nonunion in the majority resulted from an incorrect diagnosis at the time of accident, and that in the minority, even when the proper diagnosis had been made, no treatment had been carried out, often, it is true, for some justifiable reason. In a certain few, the measures used had been rather in the form of a surgical ritual and were not in any sense of the word adequate. It was astonishing to ascertain how often elderly persons, who after a severe fall were disabled on account of intense pain in the hip, were told without being carefully examined that they were suffering from a sprain, the fracture not being diagnosed until months later, usually by

3. Whitman, Royal: A New Treatment for Fracture of the Neck of the Femur, Med. Rec. **65**:441, 1904.

4. Henderson, M. S.: Ununited Fractures of the Hip, Surg., Gynec. & Obst. **30**:145 (Feb.) 1920.

another consultant. Another somewhat common story was that the physician who was called in carefully measured the legs and finding no shortening or eliciting no crepitus, diagnosed a sprain without resorting to radiography. Weeks or months later a consultation necessitated by continued disability revealed shortening of the affected leg, crepitus on manipulation, and a roentgenograph established the diagnosis of fracture. The probable explanation of this is that what was originally a more or less weakly impacted fracture broke down through inadequate fixation of the limb. *In none of the cases in this series was there a history of really proper treatment for the fracture. Good treatment had been instituted in a few instances, but for some reason it was not prolonged enough.* The patient may have been unruly, the supervision too weak, or complications, such as impending pneumonia, may have necessitated a discontinuance of treatment.

CAUSES FOR FAILURE TO APPLY THE ABDUCTION METHOD

Such a state of affairs should not be allowed to persist, and this article is an inquiry into the causes for failure to adopt the only proper method of treatment for a crippling injury. I have quoted, and used as texts for comment, excerpts from several authors. This is not done in a spirit of criticism. The quotations are offered as fairly reflecting the general attitude toward the treatment of the injury, which is notable chiefly for its lack of any general standard, either in methods to be applied or results to be expected. To illustrate this haziness, I have taken three passages from a recent monograph.[5] The author begins his article, a description of a splint he has devised, with a few home truths:

1. Experience has taught that these patients (suffering from fracture of the neck of the femur) bear confinement in bed and the presence of cumbersome dressings very badly.
2. Probably most surgeons treat these cases with sandbags and extensions. Imperfect rigidity of the parts is thus obtained.
3. Plaster bandages enclosing more or less of the trunk and the thigh are used by many.

If one were to read these statements and apply them to a patient suffering from a fracture of the shaft of the femur, what would be the impression of the quality of the treatment that that particular class of patients was receiving? Who would keep a patient suffering from a fracture of the shaft of the femur flat on his back, unable to move because of sandbags, making whatever movements he did at the cost of excruciating pain, because of the "imperfect rigidity of the parts thus obtained"? Who, if he used plaster, would use it "enclosing more or less of the trunk and thigh"? Would not the most charitable comment on such treatment be that the one who used it was a little uncertain in his mind as to the principles of the

5. Masland, H. C.: A Form of Splint Available in the Treatment of Fractures of the Neck of the Femur, Ann. Surg. **71**:501 (April) 1920.

treatment he was trying to apply? Certainly one would not be over-optimistic as to the result to be expected. On the basis of such treatment one would scarcely feel justified in making the statement that fractures of the shaft of the femur were a class of cases not adapted to the application of surgical principles, and that the surgeon in charge of such a case was to be congratulated if he succeeded in saving his patient's life. In short, one would say that the surgeon was blaming Nature for the natural consequences of his improper treatment.

Has not this been the attitude of surgeons in general for the past hundred years toward fracture of the neck of the femur?[6] Because such fractures "occur most commonly in elderly persons," and because surgeons have not had at their command means for the application of such surgical principles as they would at least attempt to apply in the treatment of any other fracture, have they not been prone to excuse their universally bad results on the ground of "lowered vitality and structural weakening of the part"?

I think it may now be conceded that authorities on the subject of fractures are agreed that the treatment of fractures of the neck of the femur by the abduction method is the only method that applies, or attempts to apply, surgical principles to that form of injury.[7]

Under anesthesia initial manual traction on the affected limb overcomes shortening. Full abduction and slight inward rotation places the fragments in apposition and restores the normal angle described by the neck of the femur with the shaft. Full abduction of the thigh on the pelvis holds the fragments in position by tension of the capsular ligaments and relaxation of the muscles whose contraction tends to produce overriding of the fragments. What is most important, it assures the mutual pressure of the fractured surfaces, which was insisted upon by Sir Astley Cooper as being essential to repair. In combination with full abduction, hyperextension of the thigh on the pelvis is an additional safeguard against the force of gravity in preventing dorsal displacement of the distal fragment (Figs. 1, 2, 3 and 4). Any one who has performed an open operation on such a fracture has had the opportunity to verify these facts by his own observation.

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6. Cotton, F. J.: Ann. Surg. **63**:366 (March) 1916.

7. Albee, F. H.: Orthopedic and Reconstruction Surgery, Philadelphia, W. B. Saunders Company, 1919, p. 600. Tubby, A. H.: Deformities, Including Diseases of the Bones and Joints, London, the Macmillan Company **1**:595-601, 1912. Judet, H.: Traité des Fractures des Membres, Paris, A. Maloine, 1913, p. 356. Scudder, C. L.: The Treatment of Fractures, Ed. 8, Philadelphia, W. B. Saunders Company, 1915, p. 422; A System of Surgery, New York, Funk & Wagnalls Company **3**:648, 1914. DaCosta, J. C.: Modern Surgery, Ed. 8, Philadelphia, W. B. Saunders Company, p. 673.

Indeed, to any one who has done so the proposition seems so simple and self-evident that he is inclined to wonder at the slow general progress that has been made toward its adoption.

I take it that the majority of surgeons are conscientiously interested in the welfare of their patients, and that we may not ascribe the lethargy of the profession toward the adoption of a new principle either to stupidity or to laziness. One is therefore bound to inquire what the further causes of such a condition may be. Having pointed out a theoretical truth, we may not yet be satisfied if almost universal human fallacies prevent its application. To use a homely illustration, we have not done our duty during a smallpox epidemic if we content ourselves with recommending vaccination. We must see that the vaccination takes. What, then, are the causes militating against the application of the abduction treatment of fracture of the neck of the femur?

Figure 1

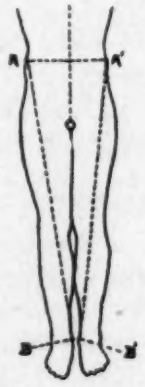

Figure 2

Fig. 1.—Attitude of the limb in fracture of the neck of the left femur.

Fig. 2.—Limbs parallel: traction on the affected limb has overcome shortening. Distance A′ B′ must equal distance A B.

The first is the general atmosphere of discouragement surrounding the patient. He, or she, is old, frequently fat, often, when first seen, already suffering from hypostatic congestion of the lungs, which after a time may become an edema causing death. There is usually a suspicious redness over the sacrum, and the nurse, or family, complains that it is next to impossible to keep the patient clean, because of the protests that she makes at any attempts to change her position. The picture is one to make the most optimistic surgeon shudder. If the patient dies untreated the death will be regarded by the family as an act of God, and frequently as one for which they are most

grateful. Such a death is commonplace. There is no community that is not familiar with the story of more or less legendary great uncles and aunts who "broke their hips," and died after varying degrees of suffering. If the surgeon applies treatment other than the generally accepted ritualistic sandbag, particularly the proverbial instrument of torture known as the "plaster cast," and the patient dies, he and his treatment will receive full blame for the death. The relatives will forget the agony of the patient, and regret only that her presence could not have been vouchsafed them for a few weeks longer. Is it any wonder, then, that a surgeon should hesitate to apply a treatment of the soundness of which he may be theoretically convinced, but which very likely he has never seen applied? Can we blame him too severely for expatiating on the notorious unwillingness of such fractures to unite, of the necessity for making the patient as com-

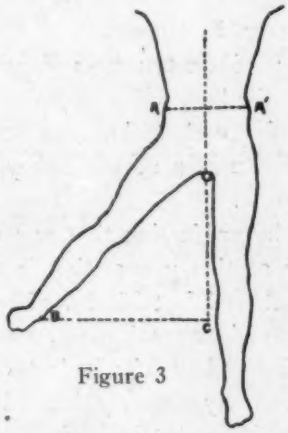

Figure 3

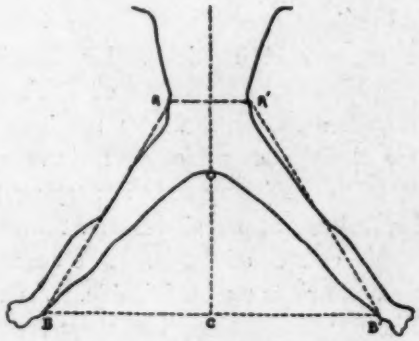

Figure 4

Fig. 3.—Abduction of the sound limb to the normal limit.

Fig. 4.—With traction maintained, the affected limb is abducted to correspond to the normal. Angle B′ O C must equal angle B O C; distance A′ B′ must equal A B.

fortable as possible, and of getting her out of bed immediately to avoid pneumonia? In such a course he is supported by tradition, and he will add to his reputation as a "sound conservative."

In this connection, I hope that I may not be accused of levity if I use the following incident as an illustration: During a clinic which I was holding on infantile paralysis in New York State I heard one physician say to another that a child in a far, outlying district had just died of "black measles." I asked what "black measles" were. "Why, Doctor," was the answer, "it's a peculiar disease we sometimes have around here. Only Dr. Smith's patients have it. He

makes the diagnosis, all the families hereabout know it's fatal, so when the patient gets no treatment and dies of 'black measles' everybody's satisfied."

The second cause for the failure to apply the abduction method is that when Dr. Whitman originally described its theory [8]—its basic anatomic principles—he also described its application by the use of the long plaster-of-Paris spica. I am seriously inclined to believe that this was unfortunate. Had the treatment been described solely by anatomic demonstration and its practice left to the individual, the grasp of the theory might not have been overshadowed and interfered with by the difficulty of a particular method of its application. So great has this confusion become that I have frequently heard the abduction treatment referred to as the "plaster treatment." It is not a plaster treatment. The means of its application are a detail, and vary according to the individual predilections of its employers. Sir Robert Jones [8] uses an abduction frame. Dr. McGlannan [9] of Baltimore includes both thighs. Dr. Scudder [7] of Boston puts the limb in a flexed position. Judet [7] of Paris uses the "gouttière de Bonnet." These are methods that are much at variance, but that are sound because those that employ them have a definite idea of what they are trying to do, and why they are trying to do it.

There is an impression current among the profession that anybody can apply plaster of Paris. The corollary of this proposition is that plaster of Paris is uncomfortable. As a matter of fact I know of nothing in surgery which requires more practice, or is more difficult, than to apply a comfortable plaster-of-Paris dressing. And of all plaster-of-Paris dressings there is none more difficult to apply properly than a long spica, extending from the axillae to the toes, in which the patient must live for a period of weeks. After having applied such a dressing, one has then to combat the obstinacy of the entire nursing profession. That profession, too, has its traditions, of which one of the most firmly grounded is that patients encased in plaster are as immovable as the Rock of Gibraltar, and must lie in changeless discomfort until they die of bedsores. Any suggestion that the patient be moved at all, much less turned on the face, is met by a gasp of horror. As a matter of fact, however, and I have even had this sheepishly admitted to me by a broken-spirited attendant, it is easier to turn a patient, even with the added weight of plaster, when turning does not cause him pain, than

8. Jones, Robert: The Oxford Surgery, New York, Oxford University Press, 1918, p. 805.

9. McGlannan, A.: Fracture of the Neck of the Femur, Surg., Gynec. & Obst. **22**:287 (March) 1916.

when every movement causes discomfort, sometimes bordering on agony.

Provided the plaster has been properly applied, it should be comfortable and strong enough to permit of the patient's being freely moved about. He should be turned on his face every day, as well as from side to side, and this constant shifting of position is the most certain preventive of bedsores.[10] Unless the nurse's hand is exceptionally large, she will find it quite possible to introduce it under the plaster and give the back its daily massage.

The objection is being continually made that the "treatment is so confining." This is quite true, but I know of no treatment of what may be called a major fracture, that is not confining. Indeed, since the war, we have capable advocates of the treatment of all fractures of long bones by traction and suspension. At a recent lantern slide demonstration of this technic,[11] I heard one enthusiastic convert remark that "Dr. Blake's demonstration had sounded the death knell of the treatment of fractures by plaster of Paris." What those who object to the confining element of treatment really mean is that they fear to try any treatment at all, and, neglecting the fracture entirely, wish to get their patients up and about as soon as possible. This attitude is perfectly reasonable provided its advocates make it clear to themselves and their patients that they are abandoning all thought of functional recovery and are preserving for them a future of permanent lameness and discomfort.

As a matter of fact, in many cases, the treatment need not be exceptionally confining. A degree of activity is possible directly in proportion to the temperament—adventurous or otherwise—of the patient. I have already said that all patients should be turned on their faces daily for a varying period, and of course all patients should have the heads of their beds raised to avoid the danger of hypostatic congestion. From this as a minimum, one may proceed as far as having the patient up and out to drive. If the splinting apparatus be properly applied, movement causes no pain, and the difficulties of movement become purely mechanical and subject to the strength and ingenuity of the patient. One woman now under treatment at her own home, after remaining at the hospital for one week, had her husband sling a pole from the head to the foot of her

10. Whitman, Royal: The Abduction Treatment of Fractures of the Neck of the Femur, with Especial Reference to the Semireclining Posture and Changes in the Attitude in Its Application to Elderly Subjects, Ann. Surg. **53**:489 (April) 1911.

11. Whitman, Royal: The Abduction Treatment of Fractures of the Neck of the Femur, Med. Rec. **97**:585 (April 3) 1920.

bed, over which a rope was passed and made fast to the leg of the plaster. With this primitive block and tackle she was able to change her own position at will. Another energetic Jewish woman, aged 64, at the Hospital for the Ruptured and Crippled, insists on being propped up in a wheel chair every day. Insane patients, who could not be treated in any other way, have as a last resort been, as it were, boxed up in plaster of Paris by the abduction method and have made perfect functional recoveries. Sir Robert Jones, indeed, goes so far as to say, "Where hypostatic troubles may be expected, plaster of Paris has advantages over any kind of splint, for the patient need not be confined to bed." [8]

To quote again from the article which is serving for my text, speaking of the application of a plaster spica upon a pelvic rest, the points of support being the sacrum and the shoulders, the author says, "The vertebra [sic] are sagging in the middle with the spines projecting more than they naturally do. This factor . . . explains the great discomfort frequently experienced and the fact that bedsores are not uncommon." If plaster, or any support, be applied in this position, the consequences to which the author alludes are to be expected. One must be most careful to guard against this by full extension of the thighs upon the pelvis, thus tilting the pelvis forward, and by manual support if necessary, or the insertion of a pad or pillow, to assure the maintenance of the normal lumbar lordosis. It is the flat back, the result of the sagging of the spine following prolonged confinement in bed, that is the cause of so much discomfort, and sciatic pain.[12] How much worse if the patient be immobilized in this position. The quotation is a perfectly justified criticism of an improperly applied support. I should imagine also that this danger of the flat back, obliteration of normal lordosis, would have to be particularly guarded against if the patient were put up with the thighs flexed on the pelvis.

A third reason for the failure of the general application of the treatment is the neglect of surgeons to carry out its elementary anatomic details. For this I confess I can find no satisfactory explanation. On one occasion, I demonstrated the application of the method on two patients to the interns of a general hospital, to their apparent understanding and satisfaction. A short time after, I returned to find two other patients to whom they proudly stated the method had been applied. The affected limbs were possibly in 15 degrees of abduction, the body portion of the plaster was so short that it dug into the

12. Goldthwait, J. E., and Osgood, R. B.: A Consideration of the Pelvic Articulations from the Anatomical, Pathological and Clinical Standpoint, Boston M. & S. J. **152**:593; 634, 1905.

patients' short ribs, causing them continual discomfort, and the plasters had both broken at the groin. At times one might really be led to suppose that surgeons do not know what abduction is. This may be evidenced by Figures 5, 6 and 7, taken from a most interesting article.[13] Abduction is a motion taking place at the hip joint and not one of the patients photographed shows any abduction whatever. They are simulating abduction by tilting the pelvis. Nor do surgeons understand why abduction is important, and they are inclined to regard the insistence on the position of full abduction as an idiosyncrasy of a faddist. Full abduction is important for these reasons: It anatomically restores the alinement of the fractured bone and assures mutual pres-

Fig. 5.—Simulation of abduction by tilting of the pelvis. The patient shows no abduction whatever. (Illustration taken from an article by another author.)

sure of the fragments upon one another. Full abduction is necessary to assure internal splinting, by forcing the proximal fragment within the acetabulum, maintaining tension on the capsule, and bringing the trochanter in contact with the rim of the acetabulum. After the bone has united the patient can walk without a limp. A normal range of abduction is as essential to a normal gait as is that of flexion or extension. The secret of the indifference displayed toward this

13. Davison, Charles: Transplantation of Bone for Defects of the Head and Neck of the Femur, Surg., Gynec. & Obst. **29**:142 (Aug.) 1919.

detail lies in the fact that surgeons are astonished and gratified to have such a patient walk at all. As a criterion of success to demand that he walk with comfort or without a limp would be regarded as chimerical.

Indeed, I am sorry to say that in the general literature on the subject, the clearest and most concise statement of why abduction is essential comes from a French author, Dr. Judet:[7]

Elle (traitement) doit viser à rétablir à la fois la forme et la fonction. Il est d'ailleurs reconnu aujourd'hui que la seconde ne peut être parfaite si la première est très defectueuse.

Fig. 6.—Simulation of abduction by tilting of the pelvis. The patient shows no abduction whatever. (Illustration taken from an article by another author.)

Treatment should aim to reestablish, at the same time, form and function; it is, however, recognized today that the second cannot be perfect if the first is defective.

Evidently in France it is not considered a "crime to disturb impaction" with a view to the improvement of function, nor is the surgeon satisfied if he has succeeded in obtaining union with the bones in unsatisfactory position.

Another cause for failure lies in improper after-treatment. As Henderson[4] says, in speaking of the cases in his series, "Good treatment had been instituted in a few instances, but for some reason it

was not prolonged enough." Here again surgeons at large do not seem willing to face the fact that weight should not be borne on the affected limb for six months after the injury. This seemingly excessive caution is due to these reasons: first, the slowness of repair of intracapsular fractures; second, the peculiar situation of the injury, the body weight falling directly across the line of fracture. I suppose the reason for not considering these facts and for the tendency to allow early weight bearing with the consequent grave risk of refracture, or the development of the deformity of coxa vara, is that to

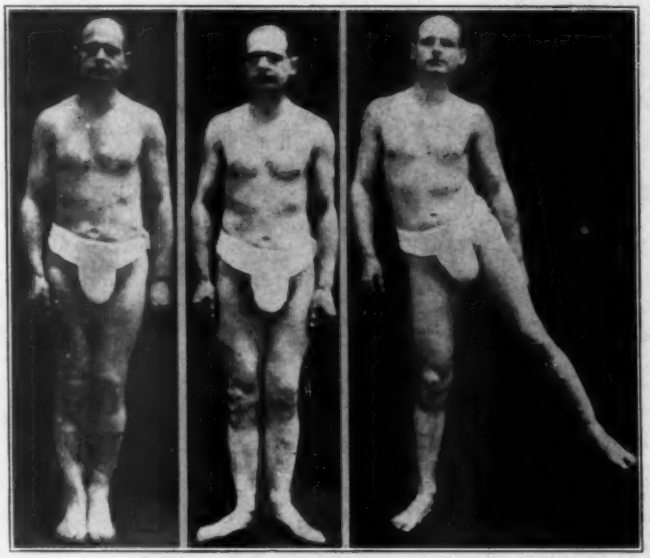

Fig. 7.—Simulation of abduction by tilting the pelvis. The patient shows no abduction whatever. (Illustration taken from an article by another author.)

most people non-weight-bearing means lying in bed, or at least getting about on crutches. This is not necessary, as the caliper hip splint will allow fairly comfortable locomotion.[14] It is possible to employ the Thomas splint for the same purpose, though it is not nearly so comfortable. Even if neither of these methods is practicable, I think the average patient would prefer six months' inaction to a permanently damaged and probably painful limb.

SUMMARY

The causes militating against the proper treatment of fracture of the neck of the femur would seem, then, to be these: (1) the tremendous force of surgical tradition to the effect that any treatment

14. Whitman, Royal: A Treatise on Orthopedic Surgery, Ed. 6, Philadelphia, Lea & Febiger, 1919, p. 555.

is useless; (2) the fact that the majority of such injuries occur in subjects that are not in any respect attractive to the surgeon; (3) lack of clear understanding of the anatomic principles of the abduction treatment; (4) the unfamiliarity of the average surgeon with the use either of plaster of Paris or the manufacture and application of other retention apparatus; (5) lack of appreciation of the necessity for the restoration of the normal form and structure of a bone if normal function is subsequently to be expected, and (6) a neglect of proper after-treatment, and the lack of insistence on detail.

In a paper such as this, and in any inquiry into failure, it is very difficult not to seem hypercritical, and I can imagine many readers laying this aside with the impression that the emphasis throughout had been on criticism, and perhaps on criticism of individuals. I hope that such will not be the case. In any walk of life there are certain facts that must be faced, and one of the most glaringly unpleasant in the surgical world is the wretched lot of the average patient with a broken hip. None of the obstacles to failure that I have enumerated is insuperable.

No one, unless self-deluded or a liar, will pretend that a certain treatment can be applied in all cases, or that all patients may be cured. What I hope to see in the immediate future is the wider recognition of the fact that a fracture of the neck of the femur is a fracture like any other fracture, even though it often occurs in subjects that one would not be anxious to treat for any complaint whatever; also that the same standards of treatment and the same judgment of results should be applied to this fracture as to any other.

Surgeons can no longer shelter themselves behind the age-long superstition to which Sir Astley Cooper one hundred years ago did not subscribe, that intracapsular fractures of the neck of the thigh bone will not unite under any form of treatment. If they do not care to apply treatment in a given case they must explain to those concerned that the character of the patient makes it inadvisable to treat the fracture, not that the character of the fracture makes it inadvisable to treat the patient.

TWELVE CASES OF THROMBOSIS OF THE CAVERNOUS SINUS

FROM A STUDY OF FIFTY THOUSAND SURGICAL HISTORIES IN THE JOHNS HOPKINS HOSPITAL *

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INTRODUCTION

Thrombosis of the cavernous sinus is a very rare condition. In 1902, Dwight and Germain [1] stated that only 182 cases had been reported, and in 1918, Smith [2] estimated the number reported as less than 300. Only eight cases were found by us in 50,000 of the surgical records of the Johns Hopkins Hospital from 1889 to 1919. This is only one case for every 6,250 patients. We are including in this report four other cases: two of these were on other services [3] in the Johns Hopkins Hospital; and we are indebted to Surgeon-General Ireland, United States Army, for the privilege of reporting two other cases (Cases 11 and 12) which were seen by Dr. Chisolm while in the army during 1918 and 1919.

REPORT OF CASES

CASE 1.—*History.*—A colored laundress, aged 40, was admitted to the Surgical Service of the Johns Hopkins Hospital, May 15, 1899, complaining of a swelling of the jaw. The personal history was negative. The present illness had begun five weeks before with toothache. A tooth had been extracted, following which the left jaw became painful and swollen. The symptoms gradually increased and the patient became restless and irritable. Four days before the patient had a chill and became drowsy. Two days before a large abscess in the left parotid region was incised.

Examination.—On admission the temperature was 104 F. and the pulse 144. Pus was draining from the incision over the left parotid region. Swelling extended to the anterior and posterior triangles of the neck. Through the incision a probe could be passed to the mastoid and posterior cervical region. The teeth of the lower jaw were loose, necrotic at the roots and bathed in pus.

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* From the Surgical Clinic of the Johns Hopkins University Medical Department.

1. Dwight, E. W., and Germain, H. H.: Thrombosis of the Cavernous Sinus; with Report of Four Cases, Including One Cranial Operation, Boston M. & S. J. **146**:456, 1902.

2. Smith, D.: Cavernous Sinus Thrombosis, with Notes of Five Cases, Arch. Ophth. **47**:482 (Sept.) 1918.

3. We wish to thank Professors Williams and Howland for the privilege of reporting Cases 7 and 10, respectively.

A few faint râles were heard at the bases of both lungs. There was a faint systolic murmur in the pulmonic area. Urine examination revealed: specific gravity, 1.022; acid; a trace of albumin; a few white and red blood corpuscles; a few hyaline and granular casts; bile +, and no sugar. Blood culture disclosed no growth. On lumbar puncture, 34 c.c. of a turbid straw colored fluid was withdrawn which contained leukocytes and a few extracellular diplococci. Cultures revealed a pneumococcus.

Course in Hospital.—There was a septic temperature ranging between 99.2 and 104.6 F. May 15, the patient was unconscious; there were marked swelling of the left jaw and edema of the face, especially of the right eyelids; right exophthalmos; edematous conjunctivae and rigidity of the neck. There was a friction rub in the right axilla. Respirations were rapid and noisy. May 17, there was increasing bulging of the right eye with edema of the surrounding tissues. May 18, coma and increased swelling of the face were present. No Kernig sign was elicited. Pulse was small and irregular; temperature was 104 F. The patient died.

Necropsy.—There was an operative wound for parotid abscess following alveolar abscess, with extension to the temporal and sphenoidal regions. There was basilar meningitis with hemorrhages into the brain, meninges and spinal cord. Cloudy swelling of viscera was present. (No note on venous sinuses of skull or paranasal sinuses.)

Etiology.—This was evidently a case of cavernous sinus thrombosis with an alveolar abscess as the primary focus, the infection involving the cavernous sinus through the pterygoid plexus. The organism was a pneumococcus. The duration of the disease was two days after the onset of eye symptoms.

Case 2.—*History.*—A boy, aged 5 years, was admitted to the Surgical Service of the Johns Hopkins Hospital on Aug. 8, 1900. The personal history was negative. The present illness began two weeks before when he fell, striking his forehead. The injury was apparently trifling. A few hours later a swelling appeared above the left eye, but this entirely disappeared in a few days. One week before, he suddenly became restless and was delirious within a few hours. There were nausea and vomiting, and for the last few days the head had been retracted. The left upper eyelid became much swollen.

Physical Examination.—On admission the temperature was 104 F., the pulse, 110. The patient was well developed, and showed a flaccid paralysis of the right leg and arm and of the left side of the face. There were restless movements of the left arm and leg and marked retraction of the head. There was marked exophthalmos on the left side with edema of the lids; the left pupil was widely dilated and immobile. The fundus showed optic neuritis; there were no hemorrhages. The right eye was normal. The nose showed a blood clot on the right side but no purulent discharge. The ears were normal. Blood count revealed: white blood corpuscles, 41,000. No specimen of urine was obtained. Culture of spinal fluid disclosed *Staphylococcus aureus.*

Operation.—Aug. 8, 1900, the left orbital abscess was incised and drained. Thick yellow pus was obtained which on culture showed *Staphylococcus aureus.* Exploratory craniotomy was performed in the left frontal region; no brain abscess was found.

Course in Hospital.—Temperature was septic in type. The day following admission, the general condition was much worse and there was edema of the right eyelids. On August 10, this edema had increased. Temperature was 107 F. There were convulsive and athetoid movements of the right arm and leg.

On August 11, the parents insisted on removing the child from the hospital. The temperature was 108.3 F. The child died shortly after reaching home. Necropsy was not obtained.

Etiology.—This was a case of left orbital abscess following injury. The infection reached the left cavernous sinus through a thrombosis of the ophthalmic vein and extended to the right cavernous sinus through the circular sinus. There was septic basilar meningitis with softening in the lower section of the pons giving rise to the crossed type of paralysis—right sided hemiplegia and left sided facial paralysis. The organism was *Staphylococcus aureus.* Duration of disease was about ten days.

CASE 3.—*History.*—A farmer, aged 32 years, was admitted to the Surgical Service of the Johns Hopkins Hospital, Oct. 31, 1909. His skull had been fractured in the right temporal region two years before. For the past two months he had been feeling bad, showing a slight elevation of temperature and occasionally complaining of left sided headache. The onset of the present illness had been sudden, occurring four days before with pain over the left eye and on the left side of the head, nausea and a chill, with a temperature ranging between 102.5 and 105 F. The pulse ranged between 60 and 80. Later there was a painful swelling in the left temporal region and cheek. Prior to admission to this hospital, the left mastoid had been explored and found normal.

Physical Examination.—On admission the temperature was 104.6 F. and the pulse 80. The patient was mentally dull and irrational. There was profuse sweating of the right side of the body. The right abdominal reflex was absent. There were enlarged submaxillary glands on the left side. Pain was felt in the neck on movement of the head. The entire left side of the face was swollen; there was slight facial weakness on the right side. There was no tenderness over the sinuses. Swelling of the eyelids was more marked on the left; the left external rectus was weak. Fundi showed no choked disk. There was fulness of the veins in the fundus of the right eye. The teeth were in good condition. The right tonsil was enlarged and covered with purulent exudate. There was an exploratory mastoid wound on the left. Blood count revealed: white blood corpuscles, 32,000. Culture of spinal fluid disclosed *Staphylococcus aureus.* No specimen of urine was obtained.

Operation.—Oct. 31, 1909, the left mastoid wound was explored. The lateral sinus was exposed and found normal.

Course in Hospital.—Temperature was continuously elevated, between 104.6 and 105.4 F. The pulse varied from 80 to 130. Nov. 1, 1909, double exophthalmos was present, the conjunctivae were very edematous, especially the right; the pupils were dilated and reacted sluggishly to light. The fundi showed no choked disk. There were marked tortuosity and distention of the retinal veins. The veins of the eyelids and forehead were distended, giving rise to a purplish color. The tongue was swollen; breathing was labored; temperature was 105 F. Tracheotomy was performed but without effect. Death followed.

Necropsy.—This revealed purulent thrombosis of both cavernous sinuses, more marked on the left side; thrombosis of the left lateral sinus; empyema of the left antrum, pus extending along the third branch of the fifth nerve through the foramen ovale to the left cavernous sinus; abscess and necrosis of the posterior wall of pharynx and purulent meningitis.

Etiology.—There was empyema of the left antrum with extension of the infection through the pterygoid plexus to the left cavernous sinus. Unilateral sweating was an interesting feature. The duration of the disease was five days. The organism was *Staphylococcus aureus.*

Case 4.—*History.*—A woman, aged 34, was admitted to the Surgical Service of the Johns Hopkins Hospital on April 28, 1910, complaining of an "infected face." The personal history was negative. The present illness began four months before with inability to open the mouth and swelling of the right side of the face. A tooth in the right upper jaw had been extracted a few days before. The swelling soon extended to the right side of the neck and the right mastoid region. Since that time the patient had been under treatment constantly. The patient had undergone five operations: (1) a tooth was extracted; the gum was incised; no pus was found; (2) an incision was made in the right temporal region followed by free drainage of pus; (3) an incision was made around the outer canthus of the right eye, three weeks later; no bone involvement was found; (4) an incision of the right side of the face from the hair line to the jaw was made one month later; (5) the right orbit was explored; no pus was found. The case was diagnosed as septic thrombosis.

Physical Examination.—On admission, the temperature was 101 F. The patient was pale and anemic. A few râles were heard in both lungs. A definite Kernig sign was elicited. Two sinuses were present in the neck just below the angle of the jaw on the right side. A large granulating wound covered with pus involved the entire right side of the face, into which opened a discharging sinus leading to a cavity under the eye. There was marked edema of the right eyelids and conjunctiva. There was a fluctuating swelling above the right ear. Examination of the mouth was unsatisfactory as the patient could part the teeth only 1 cm. The urine was negative. A culture of pus from the facial wound revealed *Staphylococcus aureus.* The Wassermann reaction was negative.

Course in Hospital.—At first, the patient improved under local treatment of the face. May 9, there was a choked disk of the right eye. May 17, fundus of left eye showed veins, full and tortuous; disk clear; media hazy: right eye, veins, tortuous. Considerable right exophthalmos was present with dilatation of veins of eyelids and forehead. May 18, there was considerable left exophthalmos.

Operation.—The right temporal region was explored. The dura was exposed and found normal. The right antrum was explored and found normal. May 19, a blood culture revealed no growth. May 24, fundi showed double choked disk. Thrombosed left lateral sinus was exposed, left jugular vein was ligated and clots from the lateral sinus were removed. May 26, the patient died. Course in the hospital extended over a period of four weeks, during which time the patient had a septic temperature ranging from 96 to 104.2 F.

To recapitulate, there was extensive infection of the right side of the face, presumably from a right upper molar tooth, which improved under local treatment. There was low grade choked disk. The right temporal region and right antrum were explored. Infection from a tooth spread to the pterygoid region and thence to right side of the face and through the pterygoid plexus to right cavernous sinus. Exophthalmos subsided somewhat, but later the condition became worse. The patient was delirious at times. There was a progressive rise of temperature. Crowe sign [4] was positive on the left side. The left jugular vein was ligated and lateral sinus explored through mastoid. (Not infected.) Thrombus extended down to the point where the jugular was ligated. Patient was unconscious and grew progressively worse. Respirations were irregular and pulse was weak. Crowe sign was positive on the right May 25 (day after left jugular ligation). Death occurred May 26, 1910.

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4. Crowe, S. J.: Bull. Johns Hopkins Hosp. **23**:321-323, 1912.

Necropsy.—This revealed the following: (1) abscess, right orbit; (2) right temporal lobe abscess; (3) thrombosis both lateral sinuses, recent in right; (4) thrombosis, anterior two-thirds of superior longitudinal sinus, and (5) diffuse purulent meningitis. No note was made on cavernous or paranasal sinuses.

Etiology.—Dental infection extending by way of the pterygoid region to the face, and from the pterygoid plexus to the right cavernous sinus. Thrombosis extended to the left cavernous sinus, thence to left lateral sinus and to the left jugular vein. Later the superior longitudinal and right lateral sinuses became thrombosed. Duration of disease was more than one month after eye signs first appeared. The organism was *Staphylococcus aureus.*

Case 5.—*History.*—A colored bartender, aged 24, was admitted to Johns Hopkins Hospital, May 27, 1910, complaining of pain and swelling of the left eye and left side of the face. He had had frequent colds; two soft chancres, two and one-half years before; otherwise the personal history was negative. The present illness began with a cold in the head two weeks before. Nine days before admission it had "settled in his eyes." Since then fever, chills, sweats, pain in the left eye and left side of the head and neck had been present. He had vomited four or five times. There was increased swelling of the left eye, which had been swollen shut for two days.

Physical Examination.—On admission the temperature was 103 F., and the pulse 114. A few fine râles were heard on both sides, front and back. There was a soft systolic murmur over the pulmonic area and retromanubrial dulness. There was marked swelling of the left eye, including particularly the upper lid, and the left cheek. The forehead and left side of the head were swollen. There was tenderness over the left mastoid. Blood count revealed: white blood corpuscles, 6,300. The Wassermann reaction was negative. Blood culture revealed *Streptococcus pyogenes.* Urine examination was positive for albumin, and revealed no sugar. Bile was present.

Course in Hospital.—There was a continuously elevated temperature of from 103 to 106 F., May 28, the left eye was more swollen; edema of the face was more marked.

Operation.—The left supraorbital abscess and the scalp were incised and drained; thin pus was obtained from both. May 29, the patient was stupid, restless, unable to swallow and suffered constant pain in the head. May 30, he died.

Necropsy.—This revealed empyema of both antrums and of the left ethmoidal and sphenoidal cells; thrombosis of the left cavernous sinus; abscess of the left orbit and left side of the face; acute purulent meningitis; extensive bilateral pneumonia with gangrene of both lungs; pulmonary edema and acute diffuse nephritis.

Etiology.—Polysinusitis with (1) left sphenoidal cells as the focus giving rise to a primary unilateral cavernous sinus thrombosis, or (2) the left ethmoid infection may have been responsible for the orbital abscess, thus resulting in thrombosis of the ophthalmic veins with extension to cavernous sinus. There was metastatic pulmonary involvement. The duration of the disease was eleven days. The organism was a streptococcus.

Case 6.—*History.*—A child, aged 4 years, admitted to the Johns Hopkins Hospital Jan. 15, 1912 (Fig. 1), had always been healthy except for three or four epileptiform attacks during the last six months. The present illness began

one week before with toothache. Two days later the right side of the face and the right eye became swollen. Two days before admission the left eye also became swollen, so that both eyes were closed. There was apparently no pain.

Physical Examination.—On admission the temperature was 103 F. and the pulse 120. The patient was rather stuporous; there was a sweetish odor to the breath. General examination otherwise was negative. The eyes showed double exophthalmos. The upper lids were edematous, so that the eyes were swollen shut. The eyelashes were matted together with exudate; the conjunctivae were edematous. Fundi showed tortuous vessels. There was no choked disk on either side. There was edema of the forehead and bridge of the nose; fulness of the right cheek and right submaxillary region. The cervical glands on both sides were considerably enlarged. The ears were negative. The teeth were dirty and the mouth foul. There was a discharge of pus in the left side of the mouth not definitely localized. Blood count revealed: white blood corpuscles, 15,000; hemoglobin, 86 per cent. Urine examination disclosed: albumin, positive; acetone, positive, and diacetic, positive.

Fig. 1 (Case 6).—Patient, tenth day of the disease, showing the marked double exophthalmos and edema of the lids.

Course in Hospital.—Temperature ranged between 101 and 106.6 F., not septic in type but a rather constant elevation. January 19, condition was unchanged. The child was perfectly conscious. There had been no vomiting since admission. January 21, the patient was more restless; the pulse was weaker; Kernig sign was negative. There was questionable rigidity of the neck. Exophthalmos was increased and lids were everted. January 23, condition had gradually grown worse; the patient was stuporous and refused food. The eyes were more prominent, the face paler and cyanotic. January 24, the pulse was very weak and rapid; respirations were rapid and labored. The extremities were cold and cyanotic. There was no Kernig sign nor rigidity. Coarse râles were heard over both lungs. The child died quietly at 6 p. m. No necropsy was obtained.

Etiology.—This was a case of septic thrombosis of cavernous sinuses, probably secondary to an alveolar abscess. There were no evidences of meningitis or pulmonary involvement. The duration of the disease was thirteen days. The organism was not determined.

Case 7.—*History.*—A housewife, aged 41, was admitted to the Obstetrical Service of the Johns Hopkins Hospital Oct. 24, 1915 (Fig. 2). Her general health had been good. She had six children and a miscarriage in 1904 at six

months. A laparotomy had been performed five years before. The patient was nine months pregnant. Two days before admission the membranes ruptured prematurely and she developed a fever. The temperature was 104 F. October 25, operative delivery was performed under ether and chloroform, a craniotomy was performed on the dead child. The patient continued to have a daily elevation of temperature between 104 and 106 F. November 3, urine examination revealed: albumin, + + +, many white blood corpuscles. Urine culture revealed an organism of the colon-typhoid group. Uterine culture was negative. Wassermann

Fig. 2 (Case 7).—Patient on third day of the disease, showing the exophthalmos and edema of the lids, more marked on the right side.

reaction was negative. During the night of November 13, three weeks after admission, the patient complained of pain over the right eye, and the following morning there was swelling around the eye with marked photophobia.

Physical Examination.—On Nov. 14, 1915, the lungs were clear. Heart examination revealed a systolic murmur at the apex, transmitted to the axilla. There was a scarred cervix uteri. No glandular enlargement was present. Exophthalmos on the right side was marked; the bulb was practically immobile. There were edema of the eyelids and photophobia. The right pupil was round and contracted. Vision was lost except for light perception. Left eye was nor-

mal. Ophthalmoscopic examination revealed on the right side an exudate on the lens capsule and a turbid vitreous. Fundus could not be seen. Blood count showed: white blood corpuscles, 20,000; red blood corpuscles, 3,700,000; hemoglobin, 70 per cent. Blood culture revealed a pneumococcus. November 15, exophthalmos had increased on the right side; there was beginning exophthalmos on the left side. The conjunctivae were injected and edematous. The cornea was steamy. November 16, there was some consolidation of the right lung, and diffuse musical râles. Pneumonia developed. November 17, deafness developed, together with signs of basilar meningitis. Kernig's sign was positive; knee kicks were + +; there were beginning opisthotonos and muscular twitchings. The Babinski sign was negative. November 18, pulmonary consolidation was increasing, meningeal signs were more marked, the heart was dilated. The temperature was 106.6 F. The patient died at midnight. The temperature was of the septic type throughout, ranging from 98 to 107 F.

Necropsy was not obtained.

Etiology.—After running a septic temperature for three weeks after delivery (accompanied by evidence of infection of the urinary tract), the patient developed signs of thrombosis of right cavernous sinus, and a blood culture taken the same day revealed a pneumococcus. Two days later signs of lobar pneumonia developed, followed the next day by signs of basilar meningitis. Pneumococcus puerperal infection is very rare. It is possible that there was a metastatic involvement of the cavernous sinuses during the course of a general pneumococcic septicemia, but it is more probable that an unrecognized right sphenoidal or ethmoidal infection extended to the right cavernous sinus. The duration of the disease was five days after the development of the eye signs.

Case 8. — *History.* — A schoolboy, aged 12 years, was admitted to Johns Hopkins Hospital, Aug. 5, 1916, complaining of swelling of right eye and right side of face (Figs. 3 and 4). The boy had had the usual diseases of childhood without complications. There had been an injury to the nose (possible fracture) six months before. Two days before the present illness he had been swimming and diving in a public swimming pool. The present illness had begun five days before admission with headache, malaise, fever and nausea. The right eye became swollen and there was diplopia. On the third day, the headache practically disappeared, but there was marked tenderness about the right eye. No nasal discharge was noticed.

Physical Examination.—On admission, Aug. 5, 1916, the temperature was 103.4 F., the pulse was 100. General examination was negative. The Kernig sign was negative. The right eye was closed, the lids swollen, red and tender. The conjunctiva was blood-shot; there was exophthalmos. The pupils reacted to light. Fundi showed full vessels on the right. No choked disk was present. The left eye was normal. The right side of the face was swollen and the glands at the angle of the jaw on the right were enlarged. On the right side of the nose there was deflection of the septum, congestion of the mucous membrane and profuse purulent discharge coming from the middle meatus. The left side was normal. Evidently there was an acute right ethmoidal infection. A roentgenogram of the sinuses revealed the frontal sinuses and antrums clear. There was trismus; the teeth could be parted only 1 cm. The right upper canine tooth was loose. The ears were negative. Urine examination revealed: albumin plus; no casts. Blood count revealed: white blood cells, 23,000. Blood culture showed no growth. The Wassermann reaction was negative. Culture of pus from right nostril showed *Staphylococcus aureus.*

Fig. 3 (Case 8).—Patient on sixteenth day of the disease. The swelling and exophthalmos on the right are beginning to subside; swelling of the left side of the head and neck is the result of the thrombosis of the jugular vein in addition to that of the cavernous sinus.

Fig. 4 (Case 8).—Patient on sixteenth day of the disease, showing the marked exophthalmos and edema of the eyelids of the left eye.

Course in Hospital.—August 6, the patient was delirious. The Kernig sign was plus. Stiff neck and general hyperesthesia were present. There was incontinence of urine.

August 7, he was noisy. There was incontinence of urine and feces. The right upper canine was extracted; pus was found at the root. Culture revealed *Staphylococcus aureus* and a nonhemolytic streptococcus. The right antrum was punctured and pus was obtained.

August 8, there was less swelling of the right eye; beginning swelling of lids and exophthalmos on the left side.

August 10, he was noisy. Eye signs on the left were increased; swelling extended down to the left side of the neck.

August 11, he was mentally bright. The Kernig sign was still positive. The neck was stiff. There was tenderness over the neck and entire head. Edema was increasing. A diagnosis of thrombosis of both cavernous sinuses, extending down into the jugular vein on the left side was made.

August 12, the right eye could be opened. The pupils reacted to light. The vision was good. Extra-ocular movements were good, although there was still some protrusion of the bulb. Ophthalmoscopic examination revealed: right veins full; disk outline hazy. There was a low grade choked disk. The left could not be seen. White blood cells numbered 27,600.

August 13, he was drowsy and irritable. The swelling extended over the left side of the head; the scalp veins were full. General hyperesthesia. Nasal discharge was decreasing. He complained of pain in the left ear. Both drums were normal. Lumbar puncture revealed: fluid; clear; tension, not increased. Cell count disclosed 1 per cm. (all mononuclears). Culture showed no growth.

August 14, the right middle turbinate was infracted. There was discharge of pus. The nasal mucosa on the right side was being shrunk several times daily by local application of epinephrin.

August 15, he complained of pain in the left knee which was tender and held in flexed position; extension was painful. Kernig's sign was negative. There was less rigidity of neck muscles. Urine examination was positive for albumin; there were many casts.

August 16, blood culture revealed *Staphylococcus aureus,* five colonies per c.c.

August 17, both eyes could be opened. Swelling and exophthalmos were less. There was no diplopia. Extrinsic ocular movements were good. The pupils reacted. Ophthalmoscopic examination revealed low grade double choked disk. The left knee symptoms were acute. There were redness, swelling and local elevation of temperature. He complained of pain in the chest. Signs of bronchopneumonia (probably a pulmonary infarction) developed.

August 19, there were swelling and fluctuation in the left frontal region and osteomyelitis of skull.

August 20, there was evident bronchopneumonia with a friction rub in the left axilla.

August 21, pain and tenderness developed in the left mastoid region. The left drum was injected, not bulging. Blood count revealed: white blood cells, 17,500. He was given *Staphylococcus aureus* vaccine, 25,000,000.

August 23, the general condition was improving. Acute suppurative otitis media developed on the left side. The left knee joint was aspirated; 25 c.c. of purulent fluid was obtained; culture revealed *Staphylococcus aureus.* Ophthalmoscopic examination revealed choked disks though less marked. *Staphylococcus aureus* vaccine, 50,000,000, was given.

August 24, roentgenograms revealed: mastoids, clear; left knee, destruction of patella, marked periarticular swelling; lungs, mediastinitis, spotty infiltration, patchy consolidation at right apex, enlarged mediastinal glands; sinuses, marked clouding of right antrum and ethmoids and left frontal.

August 25, urine examination revealed: albumin, plus; red blood cells and white blood cells and numerous granular casts. Urine culture disclosed *Staphylococcus aureus.*

August 27, there was less tenderness over the left mastoid. Otitis media was clearing up. Left knee was again aspirated. No fluid was obtained. General hyperesthesia was no longer evident. Blood culture revealed *Staphylococcus aureus,* one colony per cm. Staphylococcus aureus vaccine, 75,000,000, was given.

August 30, right antrum was punctured and irrigated; the fluid was clear. Lung signs were clearing up; there was no friction rub.

September 3, left otitis media had entirely cleared up; drum looked practically normal. There was no mastoid tenderness. Left knee was still swollen and painful. *Staphylococcus aureus* vaccine, 100,000,000, was given.

September 4, blood count revealed: white blood cells, 15,000. Blood culture revealed no growth.

September 9, there were dilated veins over the whole scalp, especially on the left side. There was swelling in the left temporo-parietal region. Ptosis and occasional internal strabismus were present on the left. Cultures of the urine revealed *Staphylococcus aureus.*

September 10, there was reappearance of swelling of eyelids on the left side (which, however, disappeared again within two days).

September 13, *Staphylococcus aureus* vaccine (autogenous, from cultures of the urine), 100,000,000, was given.

September 16, the temperature was normal for the first time, prior to this the patient had been running a septic temperature, ranging from 97 to 104 F., with apex of the curve gradually coming down.

September 18, Staphylococcus vaccine (autogenous), 150,000,000, was given. Ophthalmoscopic examination revealed: disk outline still hazy, especially nasal margins, more marked on the right side.

September 21, a fluctuating swelling in left parietal region appeared, which was aspirated. Pus was found. Culture revealed *Staphylococcus aureus.*

September 24, left knee was still swollen, a few c.c. of bloody fluid was aspirated from the joint. Blood culture revealed no growth. Urine examination revealed: albumin, 0; casts, +. Roentgen rays revealed: left knee same as above; extensive osteomyelitis of the skull involving the left side, giving a worm-eaten appearance to the bone.

October 1, several additional areas of fluctuation had appeared over the left side of the head.

October 3, there was a discharging sinus of the left knee. Urine examination disclosed: albumin, 0; casts, 0.

October 9, operation was performed under light ether anesthesia. Multiple incisions were made and sequestrectomy was performed for osteomyelitis of the skull, on the left side. The sinus of the left knee joint was enlarged and a portion of the patella was removed. The patient stood the operation well and following this showed steady improvement for the next six weeks.

Nov. 24, 1916, the patient was discharged from the hospital. The general condition was excellent; he felt strong and well. There had been no eleva-

tion of temperature whatever for the last two weeks. The eyes were entirely normal in all respects. The ears were normal. There still was a small amount of mucoid discharge from the nose on the right side. The knee was in a cast; he walked with crutches. There was one small discharging sinus near the hair margin in the left frontal region, other scalp wounds entirely healed (Figs. 5 and 6).

Comment.—An acute ethmoidal infection on the right, acquired by diving in a public swimming pool, led to a septic thrombosis of the right ophthalmic vein and right cavernous sinus. There was extension of the thrombosis to the left cavernous sinus, signs of which appeared eight days later, and into the left jugular bulb and internal jugular vein probably by way of the inferior petrosal sinus. Low grade *Staphylococcus aureus* septicemia developed with metastatic involvement of the lungs, left knee joint, kidneys and left half of skull. There were signs of meningeal involvement, but the spinal fluid was clear. Acute suppurative otitis media, left, appeared as a later complication. The pulmonary and renal signs cleared up rather rapidly. The bone metastases were treated surgically. A surgical operation directed at the primary focus was advised at an early stage of the illness, but the parents were loathe to consent and it was not urged. The local treatment consisted in irrigation of the right antrum on two occasions, infraction of the middle turbinate and frequent shrinking of the nasal mucosa on the right side to promote drainage of the infected sinuses. At first, a stock and later an autogenous *Staphylococcus aureus* vaccine was used. All the other treatment was symptomatic. Positive cultures of the infecting organism were obtained from the primary focus, the blood, the urine, the left knee joint and the osteomyelitic foci in the skull. The eye signs had practically disappeared within three weeks after onset, but there was a reappearance on the left side three weeks later, which, however, was slight and lasted only two days. The dilatation of the scalp veins gave evidence of the establishment of a collateral circulation. This was more marked on the left side, and it was on this side only that the osteomyelitic foci appeared, probably as the result of the septic emboli coming direct through this collateral circulation. Coincident with their development occurred a temporary reappearance of the eye signs on the same side. Trismus was noted in this case.

Subsequent History.—The patient returned repeatedly to the orthopedic dispensary for treatment of the knee, and to the ear, nose and throat dispensary for observation. He was last seen in May, 1920, at which time the findings were: General health was excellent; he was working but was irritable and had lost fourteen different positions during the last six months. He walked with a limp due to an ankylosis of the left knee and a slight shortening of the left leg. Roentgen-ray examination of the left knee, May 18, 1920, revealed fibrous ankylosis, the patella and femur being fixed by bone. Scalp wounds were entirely healed and covered by hair. The right eye was normal in appearance; the left showed slight protrusion of the bulb. The lids were slightly bluish and puffy. The fundi were normal; the ears were normal. The nose showed deflection of the septum, and a small amount of discharge on the right side. The right antrum was dark on transillumination, other sinuses were clear. There had been no headaches. The mouth showed a small discharging sinus in the gum above the right upper canine tooth. The throat was negative. An indefinite mass was made out in the neck on deep palpation of the left side in the region of the internal jugular vein. Crowe's sign was positive for the left side, indicating nonpatency of the left jugular vein. Evi-

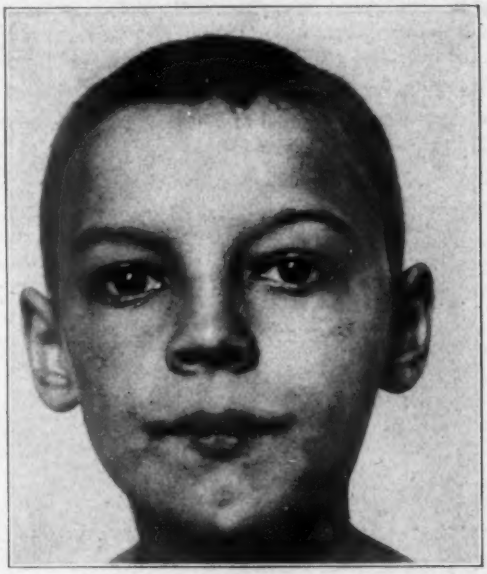

Fig. 5 (Case 8).—Patient, May 2, 1917, about six months after discharge from the hospital, showing the slight prominence of the left eye and slight puffiness of the lids.

Fig. 6 (Case 8).—Patient, May 2, 1917, about six months after discharge from the hospital, showing the scars at the points where the osteomyelitic foci in the skull were drained.

dently the thrombus in the left jugular had become organized, leading to permanent occlusion of that vein. A trifling enlargement of the scalp veins was still evident—apparently the thrombus in the right cavernous sinus had been absorbed or had become canalized, while the left cavernous sinus had probably remained occluded. The visual fields are shown in Figure 7.

Case 9.—*History.*—A housewife, aged 24 years, admitted to the Surgical Service of the Johns Hopkins Hospital, May 6, 1918, at 11 p. m., complaining of "sore eyes" (Fig. 8), had had the usual diseases of childhood, and acute tonsillitis six months before. She had "nasal catarrh." Her general health was good. The present illness began six days before when the patient noticed

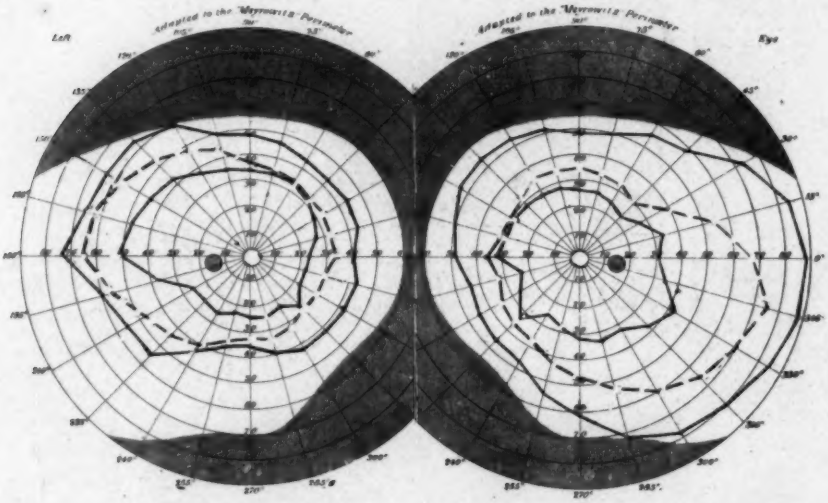

Fig. 7 (Case 8).—Visual fields, May 4, 1920, showing a definite contraction of the left field.

an inflamed area in the right side of the nose. She had fever, and the right eye gradually became affected. On the third day, there was marked swelling and discoloration of the eyelids, gradual protrusion of the eye, loss of vision and inability to move the eye. On the fourth day an abscess of the anterior nares was lanced; blood was obtained, but no pus. The left eye then became involved in the same way as the right. On the sixth day the neck became stiff. There has been headache from the first, generalized, but involving more particularly the top and back of the head. There had been fever but no chills.

Physical Examination on Admission.—The temperature was 103.4 F., the pulse 110. Both jugulars were apparently patent. There was rigidity of the neck muscles. Knee kicks were sluggish. Kernig's sign was negative. Babinski's sign was negative. The eyes showed marked double exophthalmos with edema and swelling and redness of the surrounding tissues extending to temporal region. The conjunctivae were greatly injected. The pupils were unequal; the left was irregular and reacted sluggishly. Ophthalmoscopic examination demonstrated: right, choked disk, congested and pulsating veins; left, not well seen on account of extreme photophobia. Herpes was present around the anterior nares. The left side of the nose was normal; the right side showed a crusty discharge.

The inferior turbinate was congested and ulcerated. The sinuses showed no tenderness. The right antrum was dark on transillumination; the left was fairly clear. The frontal sinuses could not be clearly seen because of swelling. Nasopharyngoscopic examination demonstrated the orifices of the posterior sinuses to be clear. The ears were negative. The throat was negative. Blood count revealed white blood cells, 19,000. Urine findings were: albumin, + + +. Blood culture disclosed *Staphylococcus aureus,* twenty colonies per c.c. On lumbar puncture 30 c.c. of turbid fluid was removed. Tension was not increased. Cell count numbered 1,500, mostly polymorphonuclears. Pellicle formed after three hours' standing. Culture demonstrated *Staphylococcus aureus.* Roentgen-ray reports were: skull showed nothing abnormal, sinuses clear.

Fig. 8 (Case 9).—Patient on the fourth day of the disease, showing the extreme exophthalmos and swelling of the eyelids.

Course in Hospital.—On the morning following admission, the right antrum was punctured and irrigated and a small amount of flaky material was washed out. On the afternoon of the same day, a right external ethmoid operation was performed.

Findings at Operation.—Ethmoid cells were filled with polypoid tissue. Culture revealed *Staphylococcus aureus.* The sphenoid was clear. A small subcutaneous pus pocket was found over a necrotic area in the lacrimal bone. May 8, the patient's condition was much worse. She was comatose; there were stertorous breathing, carphologia and rigidity of muscles of the extremities and neck. Kernig's sign was positive. Lumbar puncture revealed turbid fluid with the same characteristics as given above. At 11 p. m. Cheyne-Stokes respirations developed. Shortly after midnight she vomited and died a very few moments later. During the stay in the hospital the temperature ranged between 100 and 104 F. Necropsy was not obtained.

Etiology.—This was a case of right chronic ethmoidal infection with the development of an acute infection of the right ala nasae, which was incised. The infection traveled from the veins, draining the ala nasae to the ophthalmic vein and thence to the right cavernous sinus. A septic thrombophlebitis of the angular vein was probably responsible for the pus pocket found over the lacrimal bone. Symptoms of thrombosis of the left cavernous sinus appeared about twenty-four hours after those on the side first affected. There was an associated septic basilar meningitis and a toxic nephritis. *Staphylococcus aureus* was obtained from the right ethmoid region, the blood and the spinal fluid. The duration of the disease was eight days after onset of the superficial infection and six days after development of the first eye signs.

Case 10.—*History.*—A child, aged 22 months, admitted to the Johns Hopkins Hospital, June 23, 1918 (Fig 9), had been treated at the Harriet Lane Dispensary in June and July, 1917, for acute otitis media, cervical adenitis and

Fig. 9 (Case 10).—Patient on the fourth day of the disease: the signs are more marked on the left side as the left cavernous sinus was the first to become thrombosed.

peritonsillar abscess. She had been under observation at intervals since that time for regulation of feeding. Two weeks before the onset of the present illness she had measles, from which she recovered and had been up for several days when a swelling of the left side of the face and the left eye appeared two days before admission. The following day the swelling seemed to decrease but on the day of admission it became more marked and began to involve the right eye also. She had had fever for three days, slight cough but no vomiting.

Physical Examination.—On admission the temperature was 105.2 F., the pulse 204, and respirations, 48. She was dull and irritable. There was general glandular enlargement. The lungs showed consolidation of the right lower lobe and a marked friction rub. The left lower lobe showed the same signs to a less extent. Kernig's sign was positive. Knee kicks were not obtained. There was no stiff neck. There was marked tache cérébrale over the left parotid region. The eyelids were edematous, especially the left. Double exoph-

thalmos was present. The eyelids were red and tense. There was a small amount of purulent discharge in the conjunctival sacs; a smear made from this was negative. The left side of the face was edematous and injected, particularly in the parotid region, and the edema extended down into the neck to the left shoulder and clavicle. There was a slight nasal discharge. The ears were normal. The throat was slightly injected, otherwise it was normal. The teeth were negative. Blood count revealed: white blood cells, 23,500. Von Pirquet's sign was negative. Not enough blood was obtained for blood culture. Lumbar puncture demonstrated cloudy fluid under increased pressure. There was a film formation after standing fifteen minutes. Smears showed a few organisms suggesting pneumococci; culture, unfortunately contaminated. No specimen was obtained for urinalysis.

Course in Hospital.—June 29, the eyes were more bulging; the left lids were tense and shiny; the neighboring veins were dilated. There was a marked urticarial eruption over the entire body. June 30, the left upper arm and shoulder were markedly swollen. The left leg was held in flexion. There was loss of sensation over the left leg. Kernig's sign was more marked. July 1, the urticarial rash was much less evident during the last twenty-four hours. There was no retraction of the head. The patient died at 7 a. m. The temperature was constantly elevated during the three days in the hospital, ranging between 104 and 105 F. Necropsy was not obtained. The clinical diagnosis was acute lobar pneumonia, thrombosis of the cavernous sinuses, pneumococcus meningitis and fibrinous pleurisy. The eye signs, left, lasted five and one-half days; right, three and one-half days.

Etiology.—This was not determined. The ears, throat and face showed no possible focus. The presence of a nasal discharge would point to the possibility of a sinus infection.

CASE 11.—*History.*—A private, Q. M. C., aged 21 years, admitted to the Base Hospital, Camp Grant, Ill., at 9:30 p. m., Sept. 7, 1918, complaining of headache and a boil on the nose, gave a negative history except that his mother had died of tuberculosis four years previously. The present illness had begun four days before admission with a boil on the bridge of the nose, which was incised at a regimental infirmary. For the last three days he had severe headache, which had kept him awake every night. On the day of admission he was nauseated. There was no vomiting. He felt chilly but he had no actual chill. The temperature at 4 p. m. is said to have been 104 F.

Physical Examination.—On admission the temperature was 98.6 F., the pulse, 88. The patient appeared to be suffering with intense headache, but felt perfectly able to walk to the ward from the receiving office. General examination was entirely negative. The Kernig sign was negative. There was no rigidity of the neck. The knee kicks were equal and active. The eyes were normal in appearance; the conjunctivae were clear; the pupils were equal and reacted. The nose, ears, throat, teeth and sinuses were clear. The only positive finding was a furuncle on the bridge of the nose, about 1 cm. in diameter. This had been incised and was draining bloody, creamy pus. A culture from this pus revealed *Staphylococcus albus.*

Course in Hospital.—The patient was put to bed at 10 p. m. and given acetylsalicylic acid and codein for the pain. He vomited twice between midnight and 3 a. m. By 3:30 a. m., the temperature had gradually risen from 98.6 to 101.8 F. and the right eye was noticeably bulging and the lids slightly puffy. At 4 a.m., marked exophthalmos had developed on the right side with

edema of the lids. The conjunctiva was injected. The left eye was normal. The patient was quiet and rational but complained of intense headache. A diagnosis of thrombosis of the right cavernous sinus was made. At 7:30 a. m. the temperature was 102 F.; the patient was semidelirious. Right exophthalmos increased; there was loss of vision in the right eye. The left eye was normal. Blood count revealed: white blood cells, 19,500; polymorphonuclears, 80 per cent.; eosinophils, 2 per cent.; small mononuclears, 12 per cent.; large mononuclears, 4 per cent.; transitionals, 2 per cent. Blood culture revealed *Staphylococcus albus*. At 9 a. m., the patient was comatose. The left pupil was of pinpoint size; there was chemosis of the conjunctiva; the cornea was hazy. Edema of the right eyelid was increasing. Edema was present at the angle of the jaw and over the tip of the mastoid on the right side. Kernig's sign was positive, the neck was stiff. Knee kicks and Babinski's sign were negative. At 10:30 a. m. there was beginning edema of the left eyelids. Haziness of the media on the right prevents satisfactory examination of the fundi. On the left, the vessels were engorged; there was no choked disk. At 10:30 p. m., respirations were stertorous. Paralysis of the right arm and leg developed. Knee kicks were + + and Babinski's sign was positive on the right. The temperature was 105.8 F. (axilla). Exophthalmos had developed on the left. There was extreme edema on the right extending over the forehead and down the right cheek to the angle of the jaw. The pulse gradually became weaker and respirations became more labored and irregular until death occurred at 6 p. m., twenty-one hours after the patient walked into the hospital. The temperature and pulse gradually rose from 98.6 F., and 88, respectively, on admission to 106 F., and 175, respectively, shortly before death. The clinical diagnosis was: furuncle on the nose; septic thrombosis of the cavernous sinuses; septic meningitis, and right hemiplegia.

Necropsy.—This revealed thrombosis of both cavernous sinuses and both superior petrosal sinuses: the clot was liquefying and semipurulent. There were edema and infiltration of the tissues of the right orbit; general septicemia with embolic foci in the viscera; localized basilar septic meningitis, and a furuncle on the bridge of the nose. The paranasal sinuses, middle ears and mastoids, were clear. Cultures from the blood, cerebrospinal fluid and cavernous sinuses demonstrated *Staphylococcus albus*.

Comment.—This case was remarkably fulminating in type. Death occurred twenty-one hours after admission, at which time the physical examination was entirely negative, and fifteen hours after the first appearance of eye signs on the right. The path of infection was along the angular vein, ophthalmic vein and cavernous sinus. Seven hours after the appearance of eye signs on the right, similar signs became evident on the left. There was early evidence of meningeal involvement, with the development of a right sided hemiplegia. The right fundus was never satisfactorily examined, but the left was examined repeatedly and showed engorgement of the veins but never a characteristic choked disk and no retinal hemorrhages.

Case 12.—*History.*[5]—A private, Medical Department, U. S. Army, aged 25, was admitted to Evacuation Hospital No. 30, A. E. F., Mayen, Germany, about Feb. 23, 1919. The personal history was negative. There was no history of sore throat or trouble with the nose or ears. The present illness had begun

5. The field card of the following case is not available, and for this reason the data are given from memory and are therefore far from complete.

when routine throat cultures were taken on the personnel of the hospital unit and the patient was among those who showed a positive culture of *B. diphtheriae*, though he gave no history of ever having had the disease. He was admitted to the isolation ward.

Physical Examination.—On admission this was negative except for hypertrophied tonsils. The ears, nose and sinuses were normal.

Course in the Hospital.— The throat cultures continued to be positive for *B. diphtheriae* in spite of several injections of antitoxin and local treatment of the pharynx and tonsils. After the patient had remained in the isolation ward for more than a month removal of the tonsils was determined on.

Operation.—March 27, 1919, tonsillectomy was performed under local anesthesia (swabbing with 10 per cent. cocain and injection of 1 per cent. procain containing a few drops of adrenalin). Both tonsils were easily and cleanly removed by dissection down to the base where a Tyding's snare was used. They were not adherent and there was little bleeding. After the operation was completed the tonsillar fossae were swabbed with compound tincture of benzoin. Cultures from each tonsil gave an abundant growth of *B. diphtheriae.*

Subsequent Course.—The postoperative course was uneventful until March 30, 1919, three days after the operation, when the patient began to complain of frontal headache and developed a temperature of 101 F. On April 2, six days after operation, the classical signs of cavernous sinus thrombosis were evident, appearing first on the right side, and two days later the patient died in coma. There were never any signs of local infection in the throat and no enlargement of the cervical glands. On April 2, examination of the fundi revealed a choked disk on the right side.

Necropsy.—This revealed thrombosis of both cavernous sinuses and basilar meningitis. The jugular veins were not thrombosed. The paranasal sinuses, middle ears and mastoids were normal. Result of cultures at necropsy are not known except that the infecting organism was not the diphtheria bacillus.

Comment.—A vein or veins draining the right tonsillar fossa must have become thrombosed, and the infection must have spread from this point to the pterygoid plexus and thence to the cavernous sinus. This unfortunate case presents a rare complication following tonsillectomy, and we have been unable to find a similar one in the literature. Septic thrombosis of the cavernous sinus has, however, been reported as a complication of a peritonsillar abscess.

HISTORICAL

Cavernous sinus thrombosis was first observed at necropsy by Duncan [6] in 1821. The first clinical report was made by Vigla [7] in 1839. Other early reports were by de Castelnau and Ducrest [8] in 1846, by von Pitha [9] in 1859, and von Cohn [9] in 1860. In 1868, Herman Knapp [10]

6. Duncan, A., Jr.: Contributions to Morbid Anatomy, Edinburgh M. & S. J. **17**:334, 1821.

7. Vigla, E. N.: De la morse aiguè chez l'homme, Thesès de l'école de médecine **17**:75, 1839.

8. De Castelnau, H., and Ducrest, J. F.: Rechérche sur les abscès multiples, Paris, 1846, p. 138.

9. Referred to by Knapp, Herman: Affection des Sehorgans bei Thrombose der Hirnsinus, Arch. f. Ophth. **14**:220, 1868.

10. Knapp, Herman: Arch. f. Ophth. **14**:220, 1868.

reported a case and called attention to the symptomatology of this condition. This is one of the best papers we have found in the literature. Knapp brings out very clearly the points of differential diagnosis between cavernous sinus thrombosis, orbital abscess, and other inflammatory conditions that cause exophthalmos.

The first operation on the cavernous sinus was performed by Dwight,[1] of Boston, in 1900. It was somewhat similar to Krause's operation for resection of the gasserian ganglion. The sinus was incised, a clot removed, and a cigaret drain inserted for drainage. The patient succumbed, however, within a few hours. One month later, Hartley,[11] of New York, performed a similar and successful operation, removing from one cavernous sinus a small round-cell sarcoma which was obstructing its lumen. The patient lived for several months, but eventually died of metastases. The work of Hartley showed that the sinus could be successfully operated on, and soon several methods were devised. The different types of operation will be described later.

ANATOMY

In the adult[12] the cavernous is a paired sinus which extends along the body of the sphenoid bone from the sphenoidal fissure to the apex of the petrous portion of the temporal bone. It measures about 2 cm. in length and about 1 cm. in diameter, but is of very irregular form. It is almost quadrilateral on cross section. The lumen is greatly reduced in size: (1) by numerous trabeculae which traverse it and divide it into triangular spaces, and from which fringelike projections hang freely into the blood current, resembling on cross section the corpus cavernosum penis, whence its name; and (2), by the fact that the internal carotid artery, the carotid sympathetic plexus, and the abducens nerve transverse it. Embedded in its outer wall are these cranial nerves arranged in the order named from above downward: the oculomotor, the trochlear, and the ophthalmic and the maxillary divisions of the trigeminus.[13]

The cavernous sinus receives the ophthalmic veins (or vein), veins from the neighboring portions of the dura mater, from the sphenoidal

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11. Reported by Knapp, H.: A Case of Traumatic, at First Doubtful, Orbital Sarcoma, Followed by Aseptic Thrombosis of the Cavernous Sinus, Arch. Ophth. **29**:463, 1900.

12. In the child the cavernous sinus is represented by a venous plexus in the dura mater, which by fusion is converted into the sinus of the adult. In old age, the trabeculae diminish and the cavity is less broken up. Quain: Anatomy, New York, Longmans, Greene & Co. **2**:524.

13. Piersol: Anatomy, Ed. 1, p. 872, Philadelphia, J. B. Lippincott Company.

sinus,[14] from the pituitary body through the intercavernous sinuses, the sphenoparietal sinus, and sometimes it receives the middle cerebral vein. Posteriorly, it terminates in the inferior and superior petrosal sinuses, which empty into the jugular bulb and sigmoid sinuses, respectively. Veins pass thus from its undersurface: (1) through the foramen ovale with the mandibular division of the trigeminal nerve to communicate with the pterygoid plexus; (2) through the fibrous tissue which closes the foramen lacerum medium to communicate with the pharyngeal or the pterygoid plexus; (3) through the foramen of Vesalius, likewise leading to the pterygoid plexus, and (4), occasionally, through the foramen rotundum with the maxillary division of the trigeminus. Also a close network of veins, the carotid plexus, extends downward along the carotid artery through the carotid canal, and eventually empties into the jugular vein.

The cavernous sinuses are connected by the anterior and posterior intercavernous sinuses, the one passing in front of, and the other behind, the pituitary body. Together they form the circular sinus.

Anteriorly and superiorly, the cavernous sinus is in close relation to the optic nerve and chiasm; laterally, to the gasserian ganglion, and internally, to the sphenoidal sinus [15] and the pituitary body.

There is much difference of opinion about the order of frequency, but a study of the literature suggests that the areas from which infection may lead to thrombosis of the cavernous sinus and the venous paths to the sinus, may be thus classified: [16]

1. *Ears and Mastoid.*—The sigmoid sinus and jugular bulb are connected with the cavernous sinus through the superior and inferior petrosal sinuses, respectively. These sinuses also connect the various smaller veins of the external, middle, and inner ear, and the mastoid with the cavernous sinus.

2. *Face.*—The facial vein is connected with the cavernous sinus by many anastomoses. It anastomoses with the angular vein, the supra-orbital and infra-orbital veins, the superior and inferior ophthalmic veins, the nasal veins, the facial communicating vein, and the pterygoid plexus, all of which either directly or indirectly communicate with the cavernous sinus.

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14. Thomson, St. Clair: Causes and Symptoms of Thrombosis of the Cavernous Sinus, Ophth. Rev. **27**:294, 1908.

15. Sometimes the lateral or superior wall of the sphenoidal sinus is no thicker than writing paper, and, rarely, part of it is absent. Thus, in such cases, the cavity of the sphenoidal sinus is dangerously close to that of the cavernous sinus. Thompson, St. Clair: Cerebral and Ophthalmic Complications of Sphenoidal Sinusitis: a Contribution on Two Cases with Autopsies, Brit. M. J. **2**:768, 1906.

16. Footnotes 1, 14 and 22.

3. *Orbit and Eyelids.*—The ophthalmic vein drains the eyeball and through the angular vein the upper lid, and the palpebral branches of the facial vein drain the lower lid. The ophthalmic veins empty directly into the cavernous sinus. The palpebral veins communicate with both the angular and temporal veins.

4. *Nose and Accessory Nasal Sinuses.*—The angular vein, the nasal veins, the anterior and posterior ethmoidal veins, and some of the sphenoidal veins empty into the superior ophthalmic vein and thence into the cavernous sinus. Veins from the sphenoidal sinus may also empty directly into the cavernous sinus.

5. *Mouth and Pharynx* (including the teeth and tonsils).—Various veins of this region empty directly or indirectly into the pterygoid plexus, which is connected with the cavernous sinus through the vein of Vesalius and veins through the foramen ovale and the foramen lacerum medium.

6. *Neck and Scalp.*—The veins of these regions are connected with the cavernous sinus indirectly through the various veins of the diploe—the emissary vessels, and the occipital and posterior auricular veins.

ETIOLOGY

The three most common causes of a thrombosis of the cavernous sinus are: marasmus, trauma and infection. Marasmic thrombosis usually occurs in association with debilitating conditions at the extremes of life. The cavernous sinus in this condition is always involved after the longitudinal or lateral sinuses; it is never primarily involved.[17]

The cavernous sinus may become thrombosed as the result of an injury to the skull. When it does it is more often associated with a fracture of the base. There are a few cases reported in which thrombosis has followed a direct injury to the sinus, both from wounds through the orbit and, accidentally, during operations on the gasserian ganglion.[18]

Septic thrombosis is by far the most common form. It is nearly always secondary to a thrombophlebitis of some of the afferent or efferent veins. But it may be primary in the cavernous sinus, due to

17. Macewen, W.: Pyogenic Infective Diseases of the Brain and Spinal Cord, Glasgow, 1893, p. 245.

18. Cushing, Harvey (The Surgical Aspects of Major Neuralgia of the Trigeminal Nerve, J. A. M. A. **44**:920 (March 25) 1905) reported a case in which thrombosis of the cavernous sinus followed an injury to the sinus during an operation for resection of the gasserian ganglion, and which was not septic. The following symptoms appeared one month after the operation, suddenly, without pain and without the patient's knowledge: protrusion of the eyeball, rapidly forming edema of the eyelids, early retinal hemorrhage, and, later, blurred vision and opaque structures.

direct extension from a basilar meningitis, abscess of the postorbital space, a suppurative sphenoiditis, or suppuration of the petrous portion of the temporal bone. The bacteria most often reported are *Staphylococcus albus* and *Staphylococcus aureus, Streptococcus pyogenes,* the pneumococcus and *Diplococcus intracellularis.*[19]

PATHOLOGY

Marasmic thrombi are sterile. As a rule the thrombus is also sterile in the traumatic and operative cases. Septic thrombi, as elsewhere, begin with a phlebitis and the usual phenomena of thrombosis follow. At first, the thrombus is red and friable. It is adherent to the walls and usually completely fills the lumen. It may become purulent. The walls of the sinus are thickened and lusterless and often perforated. Usually there is involvement of both cavernous sinuses and of other dural sinuses.[20] Often there is a purulent meningitis, usually basilar. Other complications may be meningeal hemorrhages, a serous meningitis of Quincke, brain abscess and emboli to the lungs and various parts of the body, with infarction of the lungs, liver, spleen or kidneys. Usually, there is more or less infection of the neighboring vessels and nerves. Frequently there is necrosis of the sphenoid and petrous portion of the temporal bone.

SYMPTOMS

The symptoms of cavernous sinus thrombosis may be divided into three groups: (1), those due to venous obstruction; (2), those due to involvement of the neighboring nerves, and (3) those due to general sepsis. Those of the first two groups are the most important and of most diagnostic value. Those of the third group are usually dependent on an associated septicemia.

The symptoms of venous obstruction are: exophthalmos, edema of the retina and edema of the eyelids and bridge of nose. They are due to intraorbital venous congestion and distention of the tissues behind the eyeball. Usually both eyes are involved, but one before the other; the second eye, as a rule, within forty-eight hours after the first. The symptoms may almost disappear in one eye while developing in the other, and their degree may vary several times during the course of the illness. Associated with the retinal edema, one usually sees a dilatation and tortuosity of the retinal veins, retinal

19. Bacteriologic findings are frequently not included in the reports of cases, and it is difficult to determine the percentages from a small number of cases.

20. In more than half of the cases, both cavernous sinuses are involved (Dwight and Germain, Footnote 1).

hemorrhages, and, sometimes,[21] a slightly choked disk. Clouding of the media and opacity of the cornea occur within a few hours after the onset of the eye symptoms, and therefore frequently the fundus cannot be seen. Edema of the upper eyelid may be so great as completely to close the eye. When the pterygoid plexus is thrombosed, there may be edema of the pharynx and tonsil on the same side; of the skin over the mastoid and neck, when the lateral sinus and jugular vein become thrombosed, and of the face, when the facial vein is likewise involved (Fig. 3).

The cranial nerve symptoms are: ptosis, dilatation of the pupil, restriction of the movements of the eyeball, loss of vision, pain in the region of the head supplied by the ophthalmic division of the trigeminal nerve and very rarely trismus. Ptosis is nearly always due to paralysis of the oculomotor nerve, but the closing of the eye may be due entirely to an extreme edema of the upper lid. Dilatation of the pupil may be due to a paralysis of the oculomotor or stimulation of the sympathetic plexus. This varies somewhat, probably due to changes in pressure or to the inflammatory involvement of these nerves. Early in the illness, the movements of the eyeball are restricted and soon the eye is almost or completely fixed. This is due to paralysis of the oculomotor, trochlear and abducens nerves. Pain in the eyeball and supra-orbital headache are frequently early symptoms and may be quite severe. The pain is more general and increased in severity when the meninges are involved. Trismus is a rare symptom and is, probably, due to irritation of the mandibular division of the trigeminal nerve. It was observed once by Otto[22] and once by ourselves. Loss of vision is usually present, but vision may be retained even in severe cases as was illustrated by Case 8.

The symptoms of sepsis are: septic temperature, rapid, small and thready pulse, and chills and sweats. The temperature usually ranges from 101 to 106 F., with marked depressions. The chills occur early; toward the end they may become frequent and are followed by profuse sweats. The patient may also suffer from vomiting, delirium and coma.

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21. Stoker, F.: Arch. f. Augenh. **44**:105-146 (supplement number) 1901. When the cavernous sinus and ophthalmic veins are occluded a considerable amount of blood from the central retinal vein may escape through its anastomoses with other veins, and if the quantity of blood which enters through the central artery is reduced by the great increase of intraorbital pressure, it can be understood how this small quantity of blood may escape without inducing a choked disk.

22. Otto, K. (Zur Kasuistik der Thrombose der Sinus cavernosi nicht otitischen Ursprungs, Deutsch. Ztschr. f. Chir. **110**:176, 1911) thinks it was probably due in his case to thrombosis of the pterygoid plexus.

Often there are symptoms of meningitis, of pulmonary embolism,[23] of infarction of spleen, kidneys or liver, and of septic emboli in various parts of the body.

Sometimes the symptoms of thrombosis are more or less masked by those of the primary focus, especially when the infection begins in the orbit. Even when the progress of the disease is very rapid, as in Case 11, in which death occurred fifteen hours after the onset of symptoms; there may be marked edema of the lids, exophthalmos and other classical symptoms of a cavernous sinus thrombosis.

Laboratory studies on the blood and spinal fluid are of value in reaching a diagnosis. The leukocyte count is usually very high—41,000 in Case 2; 32,000 in Case 3; 20,000 in Case 7; 23,000 in Case 8; 18,000 in Case 9; 23,500 in Case 10, and 19,500 in Case 11. In two cases of our series, however, there was a low leukocyte count—6,300 in Case 5 and 15,000 in Case 6, a child 4 years of age. In Case 8 of our series, the lumbar puncture on the thirteenth day of the illness revealed a clear fluid with a cell count of one mononuclear per cubic millimeter. The fatal cases, however, usually show a cloudy fluid and a high cell count. Blood cultures are positive in the majority of cases.

DIAGNOSIS [24]

A history of chills and severe headache, a septic temperature and an exophthalmos that always begins on one side, but usually involves the opposite eye within twenty-four or forty-eight hours, are pathognomonic of thrombosis of the cavernous sinus. The temperature in cavernous sinus thrombosis, as in thrombosis of the sigmoid sinus or jugular bulb, is usually septic in character; in three of our cases, however, the fever was of the continuous type (Cases 3, 5 and 10).

The primary infection on the face may be insignificant. In Case 11, the primary focus was a small furuncle on the bridge of the nose of four days' duration. In Case 9, the primary focus was an infected hair follicle in the vestibule of the nose of three days' duration.

When only one eye is involved the following conditions may at times simulate a septic thrombosis of the cavernous sinus: erysipelas, cellulitis of the orbit secondary to an infection of the accessory nasal sinuses (Fig. 10), tumors of the orbit and arteriovenous fistula of the internal carotid artery (Fig. 11).

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23. Hampton, H. H., and Wharton, L. R.: Venous Thrombosis, Pulmonary Infarction and Embolism Following Gynecological Operations, Bull. Johns Hopkins Hosp. **31**:95 (April) 1920.

24. Posey, W. C., and Spiller, W. G.: Eye and Nervous System, Philadelphia, J. B. Lippincott Company, 1906, p. 871; also Footnote 17.

Orbital cellulitis, tenonitis and thrombophlebitis of the ophthalmic veins may be differentiated at times with difficulty from a cavernous sinus thrombosis. In the first two conditions there is often a history of previous eye trouble and more signs of inflammation in the eye than are usually found in a cavernous sinus thrombosis; also the signs of venous obstruction are less marked. The headache is usually less severe, and there are no signs or symptoms of a general septicemia or of meningeal involvement.

Fig. 10.—A case of orbital cellulitis secondary to an ethmoidal infection. The swelling and exophthalmos subsided promptly and permanently following an intranasal ethmoid operation.

Erysipelas often begins on the face or around a recent mastoid wound. It may, however, begin on the upper lid and for a few hours closely resemble a cavernous sinus thrombosis. The latter condition should always be considered as a possible complication of erysipelas. There are several cases in the literature in which the cavernous sinus became infected from a primary facial erysipelas.

A new growth in the orbit or sella turcica and an arteriovenous fistula of the internal carotid artery (a not uncommon complication of a fracture of the base of the skull) will scarcely be confused with a septic cavernous sinus thrombosis. The symptoms of a growth in this locality are gradual in onset, and there are no signs of a general infection. In the latter condition, there is often a history of injury and a bruit can usually be heard at some point over the forehead.

Thrombosis of the superior longitudinal sinus is usually a terminal complication in malnutrition or cachexia and is characterized by a cyanosis of the face, dilatation of the veins of the scalp and epistaxis.

Fig. 11.—A case of arteriovenous aneurysm of the internal carotid and the cavernous sinus, following a fracture of the base of the skull. Edema of the lids and exophthalmos developed gradually after the injury. A loud bruit could be heard above the inner end of the orbital ridge.

TREATMENT

All agree that the focus of infection if found should be as thoroughly removed as possible. But there is much difference of opinion over the best method of treating the thrombosed sinus itself. Many prefer to treat the symptoms as they arise. Others advocate prompt and radical operations on the cavernous sinus, as only 7 per cent. of these patients recover without operation.[1] The number of patients that have recovered as a result of operation on the cavernous sinus, however, is less than 7 per cent.

Chief among the various operations proposed for treating the sinus are: the temporal route of Dwight and Hartley,[25] the orbital route of Mosher,[26] the ethmoidal-sphenoidal route of Langworthy,[27] and Luc's[27] operation through the antrum on the opposite side. The temporal route of Dwight and Hartley is almost the same as far as the approach is concerned as Krause's operation for resection of the gasserian ganglion, the difference being that the dura is incised. Dwight found that an incision in one sinus will drain both. This operation has been performed by Dwight, Hartley and Ballance.[28] The one performed by Hartley was the only one that was successful. In Hartley's case the thrombosis was due to a small round-cell sarcoma and was not associated with in infection.

The orbital route of Mosher requires removal of the eye, ligation of the ophthalmic artery, removal of the inner half of the orbital plate of the great wing of the sphenoid bone and elevation of the brain before the cavernous sinus is exposed. An incision is then made in the outer wall of the sinus and the thrombus is removed with a cured. Adair Dighton[21] performed a very similar operation on one patient, but it was not successful. The ethmoidal-sphenoidal route of Langsworthy is similar to the external operation for removal of the ethmoidal cells. In addition, the anterior and lateral walls of the sphenoid are removed and the cavernous sinus opened by means of a blunt curet. In the Luc operation the cavernous sinus is approached through the maxillary antrum and the sphenoidal sinus on the opposite side. He considers the oblique angle thus obtained as very important. Robineau[27] employs temporary resection of the malar bone with trephination of the sphenoid.

Bircher[30] has reported a case in which he opened and drained the cavernous sinus by resecting the petrous portion of the temporal

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25. Footnotes 1 and 11.

26. Mosher, H. P.: Orbital Approach to the Cavernous Sinus, Laryngoscope **24**:709, 1914.

27. Langworthy, H. G.: Anatomic Relations of the Cavernous Sinus to Other Structures with Consideration of Various Pathologic Processes by Which It May Become Involved, Ann. Otol., Rhinol. & Laryngol. **25**:554 (Sept.) 1916.

28. Ballance, C. A., and Hobhouse, N.: A Case of Septic Thrombosis of the Left Sigmoid, Left Cavernous, and Left Inferior Petrosal Sinuses with a Suggestion for Treatment in Future Cases, Ninth Intern. Otol. Congress, 1912, p. 308.

29. Dighton removed most of the superior wall of the orbit from one inch in front of the sphenoid fissure backward, elevated the frontal lobe and curetted the cavernous sinus. Patient died. Dighton, A.: Cavernous Sinus Thrombosis of Nasal Origin, Med. Press & Circ. **96**:450, 1913.

30. Bircher, H.: Phlebitis des Sinus transversus, petrosus inferior und cavernosus. Eröffnung und Desinfektion nach Entfernung der Felsenbeinpyramide. Heilung. Centralbl. f. Chir. **22**:483, 1893.

bone. In this case there was an extensive suppuration of the mastoid and of the pyramid of the temporal bone, and a thrombosis of the jugular bulb and the inferior petrosal sinus. This accounts for the method of approach employed. The patient made a perfect recovery with the exception of a permanent paralysis of the facial nerve. The only eye symptoms in this case were those due to paralysis of the orbital nerves, and we do not believe that the diagnosis of cavernous sinus thrombosis was entirely justifiable. But if the diagnosis is accepted, this is the only report we have been able to find in the literature of a successful operation on the cavernous sinus for septic thrombosis.

CONCLUSION

Operation on the cavernous sinus was not attempted in any of our cases, and the percentage of recovery was 8.33. When operative treatment is contemplated it would seem that the location of the focus of infection should be a determining factor in choosing the route of approach. For instance, if the focus is in the orbit the route of Mosher might be employed, but if it is in the ethmoids or sphenoid the route of Langsworthy might be used. The failure of surgical intervention is undoubtedly due to the fact that a basilar meningitis and a thrombosis of the neighboring venous sinuses occur, as a rule, early in the course of the disease. A lumbar puncture should always be performed, for the patient may manifest the usual symptoms and signs of meningitis and yet have a clear spinal fluid. If examination of the spinal fluid reveals the presence of a septic meningitis, mere drainage of the cavernous sinus is manifestly inadequate. If, on the other hand, the spinal fluid is clear, the patient has a chance for recovery without operation.

SUMMARY OF FINDINGS

Infecting Organisms.—Total cases, 12: *Staphylococus aureus,* 5; pneumococcus, 3; *Staphylococcus albus,* 1; streptococcus, 1, and undetermined, 2.

First Sinus Involved.—Right involved first, 7; left involved first, 5, and unilateral involvement, 1.

Result.—Death, 11; necropsy, 6; no necropsy, 5, and recovery, 1.

Focus of Infection.—Paranasal sinuses, 3 (Cases 3, 5 and 8); alveolar infection, 3 (Cases 1, 4 and 6); facial infection, 2 (Cases 9 and 11); pharynx (after tonsillectomy), 1 (Case 12); orbital abscess (injury), 1 (Case 2); not determined (probably paranasal sinuses), 2 (Cases 7 and 10), and ears and mastoids, 0.

Complications.—Meningitis, 10 (all except Cases 6 and 8); pneumonia, 4 (Cases 5, 7, 8 and 10); nephritis (acute), 3 (Cases 7, 8 and 9); hemiplegia, 2 (Cases 2 and 11); sweating (unilateral), 1 (Case 3); bones and joints, 1 (Case 8), and no apparent complication, 1 (Case 6).

Duration of Disease (Determined by eye signs).—Longest, 35 days; shortest, 15 hours; average, 9 days; average (omitting long case), 6 days; average (omitting short case), 7 days; case of recovery, 3 weeks of eye signs.

Blood Culture.—Positive, 5; *Staphylococcus aureus,* 2 (Cases 8 and 9); *Staphylococcus albus,* 1 (Case 11); *Streptococcus pyogenes,* 1 (Case 5), and pneumococus, 1 (Case 7).

Negative: Two (one of these also negative at necropsy), Cases 1 and 4.

Not taken: Four (Cases 2, 3, 6 and 10).

Result not known: One (Case 12).

Spinal Fluid Culture or Smear.—Positive, 6 (one at necropsy only): *Staphylococcus aureus,* 3 (Cases 2, 3 and 9); pneumococcus, 2 (Cases 1 and 10), and *Staphylococus albus,* 1 (Case 11).

Negative: One (Case 8).

No Examination of Spinal Fluid: Four (Cases 4, 5, 6 and 7).

Result Not Known: One (Case 12).

Prodromal Symptoms.—Case 1, toothache for 5 weeks; Case 2, headache for two months, acute onset few hours; Case 3, 1 week after injury, acute onset; Case 4, toothache followed by facial infection for 3 months; Case 5, cold in head for 5 days; Case 6, toothache for 3 days; Case 7, septic temperature for 23 days; Case 8, sudden onset two days after swimming; Case 9, infection of anterior nares for 3 days; Case 10, measles for 2 weeks; Case 11, boil on nose for 4 days, severe headaches for 3 days; and Case 12, headaches and fever for 3 days, which developed 3 days after tonsillectomy.

DUODENAL DIVERTICULA, WITH REPORT OF A CASE

E. C. MOORE, M.D.

LOS ANGELES

Diverticula have occasionally been found in various parts of the alimentary tract. According to Buschi,[1] the rarest diverticula are those of the stomach; then follow those of the duodenum, pharynx, esophagus, ileum and colon. All the early cases were discovered during postmortem examinations; they were not suspected during life and were in no way considered responsible for the death of the patient. The records show they are more frequently found in the bodies of people past middle life. Wilkie[2] states that 83 per cent. are found in people over 50 years of age. They were considered rare then, as they are now. However, it should be noted that during the last five years, more and more cases have been reported, and it seems probable that they are of more frequent occurrence than they were believed to be at one time.

Buschi[1] found that in 1710 Chomel[3] had reported a case, before the Academy in Paris, of a woman, 80 years old, who had a duodenal pocket in which there were twenty-two stones. Chomel[3] mentions that two hours after meals this woman always experienced pain in the exact spot where this pocket lay. However, most authors give Morgagni[4] credit for having reported the first typical duodenal diverticulum in 1761.

ORIGIN OF DUODENAL DIVERTICULA

Two theories of the origin of duodenal diverticula have been advanced: the congenital and the acquired. Some investigators believe that the majority of cases are congenital, but that in a few instances the condition may have been acquired. Most authors subdivide congenital and acquired into true and false—a true diverticulum being one that contains all layers of a normal bowel, i. e., mucosa, submucosa, muscularis and peritoneum, and false diverticula those in which the muscularis is lacking.

It has not been possible to obtain the original manuscripts of the earlier investigators. For this reason, it has seemed best to enumer-

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1. Buschi, G.: Beitrag zur Untersuchung der Duodenaldivertikel, Virchows Arch. f. path. Anat. **206**:121-141, 1911.

2. Wilkie, D. P. D.: Duodenal Diverticula and Duplicature of the Duodenal Wall, Edinburgh M. J. **11**:219-229, 1913.

3. Chomel: Histoire de l'Acad. Royale, Paris, 1710, quoted by Buschi (Footnote 1).

4. Morgagni: De sedibus et causis morborum, Epist. 34, Par. 17, quoted by Buschi (Footnote 1).

ate briefly the principal hypotheses regarding the etiology of diverticula, accepting Buschi as authority for these. Fabricio asserts that pressure from scybala is the cause, while Morgagni [4] is inclined to believe the origin is congenital, although he does not deny the scybalum theory. Meckel [5] proved that many diverticula are remnants of the vitelline duct. Fleischmann [6] is of the opinion that diverticula lying near the common bile duct are caused by a spreading of the muscle fibers, consequently they are acquired. His opinion is shared by Roth,[7] Keith [8] and Basset.[9] Buschi asserts this argument is founded on anatomic and physiologic misconceptions. Keith [8] believes traction of the bile duct, together with general abdominal ptosis, can produce traction diverticula, while Roth [7] is of the opinion that they are caused by traction from an atrophic pancreas. Buschi [1] finds it hard to conceive how the bile duct could produce traction upon the walls of the duodenum. The fact that many an atrophic pancreas is found without the slightest trace of a diverticulum forces Buschi [1] to the conclusion that this is a far-fetched and artificial argument.

Another theory is that pulsion diverticula arise from abnormal pressure from within, when the passage of intestinal contents is hindered. According to Keith [8] this occurs when there is pressure upon the fourth part of the duodenum, or ptosis of the colon, which produces the same effect as pressure from corsets. Buschi [1] does not agree with these views, principally because the contents of the duodenum are fluid, hence the pressure exerted upon it must be less than upon any other part of the intestinal tract. The influence of the corset can only be very slight, because diverticula of the duodenum have been found twice as frequently in men as in women.

As contrasted with the views of these investigators, Letulle,[10] Gandy [11] and Marie [12] are of the opinion that diverticula of the duo-

5. Meckel: Handb. d. path. Anat. Leipzig, 1816, quoted by Buschi (Footnote 1).

6. Fleischmann: Leichenöffnungen, Erlangen, 1815, quoted by Buschi (Footnote 1).

7. Roth: Ueber Divertikel am Duodenum, Virchows Arch. f. path. Anat. **56**:197, 1872, quoted by Buschi (Footnote 1).

8. Keith, A.: A Demonstration on Constriction and Occlusions of the Alimentary Tract of Congenital or Obscure Origin, Brit. M. J. **1**:301, 1910, quoted by Buschi (Footnote 1).

9. Bassett: Duodenal Diverticula, Tr. Chicago Path. Soc. **7**: 1907, quoted by Buschi (Footnote 1).

10. Letulle: Malformations duodénales: diverticules périvatériens, Bull. Soc. anat. Paris, 1898, quoted by Buschi (Footnote 1).

11. Gandy: Diverticule duodénal congénital, Bull. Soc. anat. Paris, 1900, quoted by Buschi (Footnote 1).

12. Marie: Diverticules duodénaux périvatériens, Bull. Soc. anat., Paris, 1899, quoted by Buschi (Footnote 1).

denum are congenital. The following are the principal reasons which induced Buschi [1] to accept this theory:

1. The duodenum is the part of the intestinal canal which undergoes the most changes during fetal life, because the bile and pancreatic ducts are evolved from it.

2. Shaw's case, unquestionably congenital, in which diverticulum and at the same time occlusion of the duodenum were found in a new-born infant, proves that these diverticula are encountered in earliest childhood.

3. Duodenal diverticula are coexistent with other congenital diverticula.

4. Directly beneath the pylorus, pancreas accessorium (appendices pyloricae), or so-called duodenal culdesacs, which Meckel [5] had previously recognized, were frequently found.

5. Lewis and Thyng [13] observed that during the second fetal month in mammals there is found a rich epithelial growth along the entire course of the alimentary canal, especially in the duodenum and jejunum.

6. In most cases ulcers, tumors, stones, ascaris, scybala and scars are absent, also there are no changes in the pancreas, liver and intestine.

7. In the fetus the duodenum changes from a straight line to horseshoe shape. The objection that such diverticula are found in adults no longer has weight, because of Shaw's case.

8. Many diverticula are turned directly toward the head of the pancreas, resting therein as in a niche. Does it not seem absurd to believe that the posterior wall of the duodenum, which is the thickest and furthermore is reinforced by the pancreas, should be the very one to give way?

Buschi [1] in his three cases found no stones, tumors, scars, adhesions or inflamed pathologic condition of neighboring organs, in fact, nothing which indicated that the diverticula were acquired. In his second and third cases there was a slight inflammation in the duodenum and diverticulum, which he believed proved nothing, because in 90 per cent. of the necropsies gastro-enteritis was found, without causing any collapse of the intestinal wall. On the other hand, all the findings were in favor of the theory of congenital origin. Although Buschi [1] does not deny that an acquired diverticulum is possible, he maintains that duodenal diverticula in general and his three cases in particular were congenital.

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13. Lewis, F. T., and Thyng, F. W.: The Regular Occurrence of Intestinal Diverticula in Embryos of the Pig, Rabbit and Man, Am. J. Anat. **7**:505-519, 1908.

Early observers uniformly coincide in the belief that diverticula of the duodenum do not produce symptoms. Bauer [14] should be given credit for being the first to point out that in his two cases definite symptoms were associated with the diverticula of the duodenum.

Wilkie [2] reports three cases of diverticula of the duodenum, in two of which the diverticula were associated with grave disturbances in adjacent organs. In one of his cases, in a man, aged 58, calculi were found in the gallbladder, common bile duct and also in the cystic duct. In the second part of the duodenum, there was a curious condition of infolding of the duodenal wall and 2 inches below this there was a second similar infolding. About 1 inch above the papilla, there was an opening which admitted the thumb and which led into a sac filled with 15 c.c. of tenacious mucus. This large diverticulum was also associated with an accessory pancreas. This patient had been seized with abdominal pain fifteen hours before entering the hospital and died one hour after admission. His second case was that of a woman, aged 47, whose death was due to heart failure. Necropsy revealed an extreme degree of hepatic cirrhosis and a gallbladder containing four large mulberry-like stones. There was a large ulcer in the first part of the duodenum, on the posterosuperior wall, half an inch beyond the pylorus. In the second part of the duodenum, immediately to the outer side of the papilla of Vater, there was a diverticulum 1 inch in length, extending upward behind the duodenum. Wilkie [2] considers the first case a congenital diverticulum, and believes the diverticula in both cases offered strong presumptive evidence that there was some etiologic relationship between the diverticula and the biliary troubles.

Since the advent of the roentgen ray and the routine bismuth meal examination of all patients complaining of intestinal disturbances, our knowledge of the symptomatology of this condition has had its greatest impetus. By the fluoroscopic method it is now possible to see what is actually taking place. Thus several later investigators believe that diverticula of the duodenum are health disturbing factors at times. More and more proof to support this view is being assembled. The following are brief résumés of reported cases:

Forssell and Key [15] report a case of false diverticulum of the pars descendens duodeni in a woman, aged 41, who for a number of years had suffered from gastric distress, resembling that caused by ulcer.

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14. Bauer, T.: Ueber das Duodenaldivertikel, Wien. klin. Wchnschr. **25**: 879-880, 1912.

15. Forssell, G., and Key, E.: Divertikel a pars descendens duodeni diagnoserad medels Roentgenundersokning och operativ aflagnadt, Hygiea **77**:63, 1915.

The authors assert that this case is probably the first in which a diverticulum of the duodenum has been removed surgically, having previously been diagnosed by the roentgen ray. This patient has been relieved of the former symptoms since the operation.

Case [16] states he has observed eight cases of real diverticulum of the duodenum, occurring in the pars inferior. One of these patients, a woman, aged 45, had had pain in the lower dorsal region for six years. A definite diverticulum of the duodenum in the pars inferior, measuring 2.5 by 5 cm., was demonstrated by roentgen-ray examination. Food was retained in this diverticulum a little longer than the time it took for the stomach to empty itself. At operation, a diverticulum was found 5 cm. above the duodenojejunal junction in the third part of the duodenum, extending upward, behind and into the substance of the head of the pancreas. The gallbladder was drained, the appendix removed and the incompetent ileocecal valve repaired, but nothing was done to the diverticulum because of its intimate connection with the pancreas and because of its proximity to the superior mesenteric vessels. Six months after this operation the patient was still free from the severe pain.

Some interesting questions arise in connection with this case: Was the chronic pancreatitis caused by the faulty drainage of a diverticulum of congenital origin, and if so, why have the symptoms disappeared without the removal of the diverticulum? Or is this an acquired diverticulum secondary to a pathologic condition of the gallbladder and pancreas?

Basch [17] reports a case of a diverticulum of the duodenum in a woman, aged 36, in whom the location of the diverticulum and the roentgenogram and operative findings were very similar to those reported by Forssell and Key.[15] This patient had suffered for many years from symptoms suggestive of duodenal ulcer. At operation, the first and second portions of the duodenum were found to be congested and bound together by numerous fine adhesions. A large diverticulum, measuring about 3 by 4 cm., the base of which was about 3 cm. in diameter, was found at the angle between the second and third portions. The diverticulum was excised at its base, and after closing the resulting opening, a typical posterior gastro-enterostomy by suture was performed. At the time Basch wrote his article the patient showed a decided improvement in her digestion. Basch considers this case a congenital diverticulum of the duodenum.

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16. Case, J. T.: Roentgen Observations on the Duodenum with Special Reference to Lesions Beyond the First Portion, Am. J. Roentgenol. **3**:314-326 (June) 1916.

17. Basch, S.: Diverticulum of the Duodenum with a Report of a Case Diagnosed During Life and Successfully Operated on, Am. J. M. Sc. **153**: 833-841 (June) 1917.

Ritchie and McWhorter [18] report a case of diverticulum just below the pylorus on the lower side of the duodenum in a woman, aged 32. This diverticulum was 5 cm. in length and 3.5 cm. in diameter, egg-shaped, with a narrow isthmus that easily admitted the tip of a finger. A large duodenal ulcer was nearly opposite the opening of the diverticulum. The operation performed consisted of partially invaginating the wall of the sac by plication. The scar of the ulcer was invaginated with a few Lembert sutures and a typical short loop posterior gastroenterostomy was performed. In a letter from Dr. Ritchie, dated June 1, 1920, he said that all symptoms referable to the stomach had disappeared. The woman asserted that she was perfectly well and looked it, having gained about 30 pounds. In this case the history shows that as far back as the patient could remember she had had similar periodic abdominal pains with nausea and vomiting, several times accompanied by jaundice. For the last two years, the symptoms had become more frequent and aggravated. The fact that the history dated from early childhood suggested to the authors the possibility of a congenital origin. However, the explanation which they consider fits their case best is that a spastic incisura as well as a relaxation of the duodenum with ptosis predisposed to the development of a diverticulum.

When Carman [19] states that either true or false diverticula result occasionally from duodenal ulcer, true diverticula in less than 1 per cent. of the total number of ulcers, he certainly must be referring to ulcer diverticula only. It seems to me that Ritchie and McWhorter's case and the one that I am reporting are entirely different from those described by Carman.[19] In this connection it would seem appropriate to draw especial attention to Wilkie's [2] second conclusion, which expresses my views so perfectly that I quote it verbatim:

"In the first part of the duodenum three types of diverticula are met with, the congenital, the ulcer diverticulum, and the acquired wide-mouthed diverticulum of the duodenal vestibule."

Considerable confusion seems to have arisen from designating all of them simply as diverticula, whereas an ulcer diverticulum presents a totally different roentgenogram from that produced by a congenital diverticulum. Case [16] points out this distinction in his article when he says, "One occasionally finds a sort of diverticulum associated with the scar of duodenal ulcer, or with pericholecystic adhesions, but these occur in the first portion of the duodenum."

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18. Ritchie, H. P., and McWhorter, G. L.: Duodenal Diverticula, with Report of a Case Associated with a Duodenal Ulcer, Surg., Gynec. & Obst. **25**:485-489 (Nov.) 1917.

19. Carman, R. D.: The Roentgenologic Diagnosis of Duodenal Ulcer, Am. J. Roentgenol. **3**:252-256 (May) 1916.

Siegrist [20] reports a case of false acquired diverticulum of the duodenum in a man, aged 48. At the age of 23 this man experienced an abdominal traumatism which was followed by an epigastric hernia. A radical operation was performed upon him. Twenty-five years later during an exploratory laparotomy a diverticulum of the duodenum was found implanted on the second part of the segment of the intestine in the region of the ampulla of Vater and united by adhesions to the head of the pancreas. The diverticulum was removed and the patient recovered. The author is of the opinion that the diverticulum dated back to the traumatism.

REPORT OF AUTHOR'S CASE

History.—A man, aged 58, complained of a dull, heavy, dragging pain in the abdomen, 1 inch to the right and 2 inches above the umbilicus, which came about three hours after eating, and which was worse in the afternoon. He found relief in lying down, but not from taking food. Occasionally he had a sour stomach, but this condition was not constant, and he secured relief by taking soda. The trouble dated back seven years, beginning with a distress immediately after eating. This had lasted about three weeks, when it disappeared. Following this he was well for one year when he developed a bloody diarrhea with much mucus. This was diagnosed mucous colitis and treated, but without improvement. While being treated for this trouble, he was suddenly seized one day while in the physician's office with severe agonizing pain in the epigastrium, so severe that hypodermics of morphin were required. The severe pain lasted about twenty hours. There was neither fever nor vomiting. Following this, the abdomen, and especially the epigastrium, was acutely tender for about two weeks, necessitating his remaining in bed. The condition was diagnosed appendicitis. From then up to the present time, the gastric symptoms had gradually increased in severity, but there had been no regularity in time of onset.

Physical Examination.—The patient was a fairly well nourished man, with no general glandular enlargement. The mouth and teeth were in fair condition; the heart and lungs showed no pathologic condition. The blood pressure was 140, systolic; the temperature was 98.6 F., and the pulse 88. No tumor masses were felt in the abdomen, but there was a definite tender area in the upper abdomen just above and to the right of the umbilicus. No impulse was felt in the inguinal rings. Rectal examination revealed a small, soft prostate. The reflexes were normal. Urine examination disclosed: specific gravity, 1.020; acid; albumin, +; occasional red blood corpuscles and a few calcium oxalate crystals. The Wassermann test was negative. The stool was negative for blood.

Diagnosis.—The roentgen-ray examination in this case was the determining factor in the diagnosis. Dr. Frances C. Turley, our roentgenologist, made the following report, Nov. 13, 1916:

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20. Siegrist, H.: Duodenal Diverticulum in Man of Forty-Eight, Cor.-Bl. f. schweiz. Aerzte **49**:47 (Jan. 11) 1919; abst. Inter. Abst. Surg. **29**:15 (July) 1919.

The esophagus is negative. The stomach has good tone. The peristaltic waves are deep and pass out rapidly on both curves to the pylorus. There are no gastric filling defects. The pylorus does not open at once, although there is no marked pylorospasm. After a few minutes' observation the pylorus opens, the bulb fills, but is somewhat irregular in outline, and is situated more posteriorly than is normally the case. There is no tenderness over the bulb on palpation. About ten minutes after ingestion, with the patient in the left right oblique position on the horizontal fluoroscope, a large diverticulum on the anterior surface of the second part of the duodenum is seen to fill out. If the patient is asked to locate the area of greatest distress, he points each time to a point directly over the site of the diverticulum. At the four-hour observation

Fig. 1.—Condition, Nov. 13, 1916, before operation. Diverticulum of the duodenum projecting from the anterior surface of the second portion of the duodenum.

the stomach is almost empty, the diverticulum is still distended. The head of the opaque column has almost reached the splenic flexure. The cecum is freely movable, the appendix is not visualized. At the twenty-four hour observation the cecum, ascending and transverse colon contain opaque contents, the descending colon is free from shadows, the sigmoid and ampulla are opaque. The appendix is visualized, is quite long and is irregularly filled. Summary: Stomach, negative; duodenum, bulb irregular in outline, second part shows a large diverticulum on the anterior aspect (Fig. 1).

Operation.—Nov. 20, 1916, a right oblique supra-umbilical incision was made. When the duodenum was exposed a large diverticulum, 2.5 cm. by 3.8 cm., was seen on the anterior wall of the lower end of the descending portion of the duodenum. The diverticulum was more or less flattened out over the anterior surface of the duodenum by thin weblike avascular adhesions. After

the diverticulum was dissected free, it filled out with gas like a toy gas balloon, being almost spherical and 6 cm. in diameter. It had a small pedicle, the opening into the duodenum being about 1 cm. in diameter. About 2.5 cm. distal to the pyloric ring on the anterior surface of the duodenal wall was an old scar of a healed duodenal ulcer, not producing obstruction. The pedicle of the diverticulum was clamped and the diverticulum amputated. The stump was treated in a manner similar to that used in the case of an appendix stump. This, however, caused considerable stenosis of the duodenum and a posterior gastro-enterostomy was performed with the loop turned to the right instead of the left because of the position of the jejunum.

Pathologic Report (by Dr. R. B. Hill).—Gross Description: The specimen is an intestinal pouch, 2.5 cm. by 2.5 cm. by 1.5 cm. in diameter. The surface is rather irregular and is covered by a smooth layer of peritoneum. The mucous membrane is smooth and velvety in appearance and the valvulae conniventes, while present, do not stand out prominently. The wall measures 2.5 mm. with no irregular thickenings. Microscopically, sections show the mucous membrane to be made up of typical villi covered by a single layer of columnar epithelium, between which are characteristic crypts of Lieberkühn. Scattered through the stroma are many eosinophils and occasional polyblasts. Two or three solid masses of lymphoid tissue are seen just above the submucous muscular layer. The muscularis mucosae is very much thickened and is infiltrated with occasional eosinophil cells. There is no true muscular layer; however, occasional large irregular masses of bundles of muscle fibers are seen. The serous covering is somewhat thickened and has numerous eosinophils and occasional small round cells scattered through it. The blood vessels are quite large and seem to be distended. The diagnosis is: true duodenal diverticulum showing a moderate degree of chronic inflammation.

COMMENT

On June 1, 1920, this patient called on us at our request in order that we might determine his present condition. He stated that he had been absolutely relieved of all the former gastric symptoms. A gastro-intestinal examination made by Dr. Turley on June 2, 1920, resulted in these findings:

The esophagus was negative. The filled stomach had good tone, peristalsis began at once, the opaque meal passed the pylorus and gastro-enterostomy opening at once. The greater part passed the gastro-enterostomy opening. There was no tenderness over either opening. There was no hindrance to the passage of the opaque meal along the duodenum or jejunum. The gastric motility was about five hours. The ascending colon was one-half filled in five hours and was movable. The terminal ileum was movable from the colon and normal in appearance. The appendix was not seen (Fig. 2).

The interesting features of this case are:

1. A large diverticulum was found on the anterior aspect of the second portion of the duodenum.

2. A scar of an old healed duodenal ulcer was present on the anterior wall of the first part.

Fig. 2.—Stomach and duodenum, June 1, 1920, showing bismuth passing through stoma and duodenum.

3. All symptoms seemed referable to the diverticulum.

4. The diverticulum was amputated, our case being the fourth on record in which this was done, so far as we have been able to determine.

It would seem from our observations and from a study of the literature on this subject that ours was a case of congenital diverticulum. As a rule, persons having these congenital defects experience no untoward symptoms. However, occasionally symptoms do occur, probably caused by faulty drainage. Faulty drainage may produce irritation of the mucous membrane, and this may furnish a good field for bacterial growth, resulting in inflammatory changes which produce acute symptoms. Latarjet [21] is of the opinion that it seems only safe to say that nearly all diverticula of the duodenum are of congenital origin. and that mechanical or inflammatory factors cause the symptoms or direct attention to them.

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21. Latarjet, A., and Murad, J.: Les diverticules de l'intestin grêle, diverticule de Meckel excepté, Lyon chir. **11**:425-460, 1914; abstracted.

LATE RESULTS IN AUTOGENOUS INTERNAL BONE SPLINTING

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In the development of bone-grafting procedures during the last ten years, no standard method has been adopted. This is quite natural since each operator prefers the method which has given him the best results, and it cannot be asserted that any one technic has demonstrated its superiority in all cases. It is improbable, furthermore, that standardization ever will be effected, since aside from fundamental principles each case is a law unto itself. This is particularly true of the wounded soldier with pseudarthroses and more or less loss of bone substance. Many of these patients are veritable surgical derelicts having passed through prolonged infections in a number of hospitals. When they finally present themselves for definitive treatment, foreign bodies may be present, the muscles may be atrophic or replaced by cicatricial cords, the nerves may have suffered a concomitant injury, the normal skin covering is deficient, the scar tissue is always the site of a latent infection and the general resistance is much impaired. A number of articles have been published giving the immediate results of operation, and it has been assumed that with a primary union and a smooth postoperative convalescence of several weeks, the involved bone has been restored to health and the neighboring joints to functional activity. In our experience such has not always been the case, and it is believed that the later results in a series of such cases, in which operation was performed consecutively without selection, would form a contribution of some value to the general fund of knowledge.

The patients whose cases are here presented were operated by us during the month of August, 1919, at United States Army General Hospital No. 30, Plattsburg, N. Y.[1] Prior to June 15, 1919, this hospital had been used for psychiatric cases. At this time, with the closure of United States Army General Hospital No. 10, at Boston, and the base hospital at Camp Devens, we received the residue of their surgical cases in the form of cases of ununited fractures and pseudarthroses which could not have previously been brought to the point of

1. Col. J. C. Gregory, M. C., U. S. Army, commanding.

operation. Our results are consequently representative of those which may be obtained in a military hospital rapidly adapted to a new line of work at a time when morale is considerably impaired by a growing desire on the part both of officers and patients for an early demobilization.

GENERAL OBSERVATIONS

To eliminate some of the factors which make these late cases of bone repair a challenge to any surgeon, it has been found advisable to wait a sufficient length of time after the original infection for the tissues to establish a resistance and for the absorption of any remaining foci of infection. Among the British surgeons, it was the practice to wait at least a year after complete healing before attempting bone repair. In our cases the time intervening between the time of healing of the original wound and the operation was from three to thirteen months. Two were performed at three and one-half months. Of the three cases in which postoperative infection occurred, however, only one was a case of three and one-half months' duration, and in the other two the time intervening in each had been eleven months.

In the primary treatment of the lesion in most cases, there had been a fairly thorough débridement. Without going into the much debated subject of total esquillectomy as opposed to conservative débridement, it may be said that those cases in which some bone fragments were left between the bone ends proved to be better suited for autogenous splinting because of the support furnished by the intervening framework of callus formed on the fragments. These fragments were originally loose, but were attached to the main fragments by periosteum, those without attachment to periosteum or other soft parts having been discharged as sequestrums.

Exposure of the fragments and preparation of the field for the reception of the bone splint was the most tedious part of the operation. The ends of the fragments were usually attenuated, and no plane of cleavage between scar tissue and bone could be found in this location. Instead of commencing at the end of a fragment, it was consequently found more expedient to attack the shaft at a short distance from the end where the more normal tissues permitted easier approach to the periosteum. The adherent scar tissue was then dissected and peeled from the periosteum toward the end of the fragment. In one case in which a pocket of infection was discovered, the operation was carried no farther than exposure of the fragments, the bone grafting operation being postponed to a later date. There was but one such case encountered in our series, and this case is not reported here because we did not have an opportunity to perform the final operation. The English adopted this two-stage

procedure in all cases. Albee [2] employs a course of massage preceding the operation in order to bring out latent infections. All of our patients had had one or more courses of massage and many of them had undergone physiotherapy.

The problem of reduction of the fragments arose in only a few cases since loss of bone substance with an interval of soft parts was the rule. In the few cases of overriding fragments the reduction was directly effected by means of a strong lever. An automobile tire iron served very nicely as a lever for the femur.

These cases in which there were attenuated fragments and a lack of sound cortex were not suitable for inlay sliding grafts, and the method of application of the bone splint was consequently a matter of necessity rather than of election in most instances. The superiority of the autogenous over the heterogenous bone splint has been so well established that this phase of the subject needs no discussion. In all cases, the bone was taken from the cortex of the patient's tibia. A combination of the intramedullary and cortical methods was used in five cases, the cortical in two, and the intramedullary in nine. Three methods of holding the splints in place were used: tight wedging, ligatures and bone pegs. Intramedullary splints were made to fit tightly enough to prevent slipping, and in most cases they needed no other fixation. Once, after an intramedullary graft was inserted, it was found necessary to put a bone peg through the cortex of the fragment in order to fix the graft and hold it in place. Bone pegs and screws furnish a good means of fixation for cortical bone splints if three or more screws were used. The minimum number of three is necessary to supply the three essential points of a lever: fulcrum, power and weight. Henderson,[3] Ryerson [4] and others have recently been using screws previously prepared from beef bone. Ligatures of kangaroo tendon or strong catgut were used for lashing splints to the fragments in our cases of cortical and corticomedullary splints. Grooves were cut in the cortex, or what remained of it, to receive the tibial transplant. The ligatures were then swung around the entire fragment and tied securely over the splint, which gave good bony contact and sometimes firm fixation. Albee [2] uses kangaroo tendon which he puts through drill holes in the cortex. He states that this is absorbed in about forty days, and that the slight friction which it allows between the host bone and the graft stimulates the formation of callus.

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2. Albee, F. H., and Weigel, E. P.: Restoration of Loss of Bone, J. A. M. A. **74**:589 (Feb. 28) 1920.

3. Henderson, M. S.: The Use of Beef-Bone Screws in Fractures and Bone Transplantation, J. A. M. A. **74**:715 (March 13) 1920.

4. Ryerson, E. W.: Intramedullary Beef-Bone Splints in Fractures of Long Bones, J. A. M. A. **73**:1348 (Nov. 1) 1919.

Postoperative care is of the utmost importance. In some of our cases of lesions of the humerus we had ready a plaster splint extending from the supraclavicular region down along the posterior aspect of the arm, elbow and forearm to the hand. This was molded over the shoulder and arm previous to the operation, and was dry and hard, ready for application after operation. Except in those cases in which the lesion was in the upper end of the humerus, the arm was bound to the chest in plaster over the molded plaster cast. In the three cases in which the bone splint was inserted with one end in the head of the humerus and the other in the shaft, the arm was put up in abduction at about 120 degrees (external cephalic angle), and was supported by a plaster cast with a wire triangle reinforcement under the humerus and against the chest. Cases of injury to the forearm were put up with the elbow at a right angle and in the neutral position of 90 degrees rotation. Plaster casts were used to maintain this position. The thigh and leg were immobilized in a cast when the operation was performed on these parts. Casts were left on seven weeks as a minimum time for the cases of injury to the humerus. All our patients wore a support of some kind at the time they passed from our supervision from one to nine weeks after operation.

The bones repaired were: radius, 5 cases; humerus (shaft), 4 cases; humerus (surgical neck), 3 cases; ulna, 2 cases; femur, 1 case; and fibula, 1 case. A splint graft is not absolutely necessary for the fibula since after a loss of even several inches the leg recovers with good function. For pseudarthrosis of the upper end of the radius or lower end of the ulna, operation is not necessary since practically no loss of function results from these lesions.

Three patients developed postoperative infection; one due to disturbance of the external fixation and dressing on account of hemorrhage as mentioned below; the other two, presumably due to latent infection in the scar tissue.

REPORT OF CASES

Case 1.—A man, aged 23, sustained multiple wounds from a high explosive, which included a flesh wound of the left shoulder which speedily healed, and gave him slight discomfort only when he hyperabducted the left upper extremity. The main lesion was a perforating wound of the right shoulder, a shell fragment having entered the posterior aspect, 1 inch below the greater tubercle, making its exit at a point opposite on the anterior aspect, and producing a compound comminuted fracture of the surgical neck of the humerus. Within a few hours débridement was performed under ether in a field hospital. Three months later, numerous sequestrums were removed, and the wound healed one month subsequently. When first seen by us, the extremity was useless and had been on an aeroplane splint for one month. Nonunion was present although there was some callus. The wound had been healed for seven months

and the parts had received prolonged massage and physiotherapy. An intramedullary splint was inserted at the site of the fracture, the bone being obtained from the cortex of the right tibia. The existing callus was not disturbed more than was necessary to expose the ends of the fragments, and no internal fixation was employed other than a tight fit. A splint was placed on the outer side of the humerus and the extremity was held to the side by plaster bandages. Sutures were removed on the eighth day, and both arm and leg wounds were found to be clean. On the tenth day, the arm was fixed in slight abduction by a plaster cast. Twenty-four days after operation, there were two small discharging sinuses in the arm incision. When the hospital closed, eight weeks after the operation, one sinus was still present and the extremity was supported only by a Velpeau bandage. When seen by one of us, fifteen weeks after operation, the sinus had been solidly healed for some time. The soldier could hyperabduct and circumduct the extremity, and his musculature was regaining its tone (Fig. 1). Seven months after operation he reported his condition as "good."

Case 2.—A man, aged 26, sustained a gunshot fracture of the upper third of the left humerus. A sniper's bullet entered the anterior aspect of the arm just above the insertion of the deltoid and made its exit below the left scapula. After a first aid bandage had been applied he lay in a trench for two days without food or drink when he was taken prisoner. A plaster cast was then applied with his arm at right angles to the body. The wound healed without further attention and the cast was not removed until eight weeks later. At this time the arm was supported by a sling, but he was unable to move the upper extremity. About this time he had a severe attack of influenza. After his convalescence, he passed through several hospitals where he received massage and physiotherapy. At the time of operation the wound had been healed for nine months, but the upper extremity was useless. A splint of tibial cortex was forced in the upper fragment and fixed in the medullary canal of the lower fragment by a bone peg passing through both the fragment and the graft. This peg was necessary since a tight fit could not be secured and the graft showed a tendency to rotate in the lower fragment. There was no postoperative infection. Three weeks after operation, a fresh cast was applied with the arm in abduction, being held in this position by a wire gauze triangle in the axilla. Eight weeks after operation the graft had slipped down into the humeral shaft. Six months after operation, the soldier wrote, "No union. Need another bone graft."

Case 3.—A man, aged 23, sustained a gunshot fracture of the upper third of the right humerus resulting in nonunion with a 2 cm. loss of substance. Seven months after the wound had healed, a splint of tibial cortex was tightly wedged into the medullary canals of both fragments. This patient had taken great care to keep up the muscular development of the injured extremity and during his convalescence did all he could to "hurry" his cure. The sutures were removed in eight days and the wound was dry and its condition was all that could be desired. About three weeks after operation, there was persistent hemorrhagic oozing from both arm and leg wounds for several days. Seven weeks after operation, when he passed from our observation, the union seemed to be firm. The arm was splinted and in the Velpeau position (Fig. 2). When last heard from, seven months after operation, there was no union and a second operation was performed shortly thereafter.

Case 4.—A man, aged 23, sustained a gunshot fracture of the middle third of the left humerus. The wound healed in eight weeks, but the fracture did not

unite. After the wound had been healed eleven months and massage and physiotherapy had been employed, a piece of tibial cortex was wedged in the medullary cavity of the lower fragment and laced in a cortical groove in the upper fragment. The wounds were clean when the sutures were removed. When he passed from our observation, there was still some motion present at the site of fracture. When last heard from, seven months after the insertion of the bone splint, he reported that his arm was the same as before operation.

Fig. 1 (Case 1).—Left humerus, twelve weeks after operation.

CASE 5.—A man, aged 27, sustained a gunshot fracture of the middle third of the right humerus. The wound had been healed eight months and an unsuccessful bone graft had been performed four months previously. A splint of tibial cortex was tightly wedged into the medullary canals of both fragments. Five weeks later he was transferred to another hospital, the alinement was good, and the extremity was in a cast. A roentgenogram, taken

eight weeks later, showed that the splint was broken at the site of fracture (Fig. 3). Seven months after operation he wrote that "the graft did not take."

Case 6.—A man, aged 27, sustained a gunshot fracture at the junction of the middle and lower thirds of the left humerus. Débridement was performed within twenty-four hours, and seven days later the bone was plated and a Thomas splint applied. The plate remained in situ six weeks when it

Fig. 2 (Case 3).—Left humerus, two months after operation.

was removed, evidently on account of infection. The wound healed eight weeks subsequently, and when first seen by us there was a pseudarthrosis of the left humerus with a loss of bone of 1.5 cm. Three and a half months after the wound had healed and after the usual course of massage and physiotherapy, a piece of tibial cortex was tightly wedged into the medullary canals of both fragments, a molded splint was applied and the arm bandaged to the

side (Fig. 4). As a result of latent infection, the splint became loose and was removed five months after its insertion. When last heard from the patient was being treated expectantly.

Case 7.—A man, aged 23, sustained a gunshot fracture of the surgical neck of the right humerus, a piece of shrapnel having entered through the triceps and having made its exit through the biceps at the upper third of the arm. The wound was badly infected and was treated with surgical solution of chlorinated soda (Dakin's solution) from the ninth to the seventeenth week, when the wound had completely healed. During this period the greater portion of the head of the humerus had been removed from the wound as sequestrums. A splint of tibial cortex was wedged into the remaining shell of the head of the humerus above, and into the medullary canal of the lower fragment below.

Fig. 3 (Case 5).—Right humerus, three months after operation.

During the next few days, the fixation had to be disturbed several times on account of continued oozing from inside the humeral head. This led to movement of the upper end of the splint which caused more hemorrhage. The remainder of the head, consisting only of the cartilaginous surface poorly supported by a thin layer of osteoporotic bone, made it impossible to secure solid fixation. A vicious circle of hemorrhage, dressing, movement and more hemorrhage supervened which terminated in sepsis and the abandonment of the case to treatment with surgical solution of chlorinated soda.

Case 8.—A man, aged 22, sustained a gunshot wound of the lower third of the left forearm with a compound comminuted fracture of the radius. The left hand was abducted so that the ulnar styloid was 3 cm. below the radial. Five cm. of the radius was missing, and rotation of the forearm was impossible. The patient had received massage and physiotherapy, and the wound had been healed eight months. An autogenous splint from the left tibia was wedged into a hole drilled in the lower fragment and into the medullary canal of

the upper fragment (Fig. 5). Convalescence was interrupted, and when last heard from, six and a half months after operation, the patient reported the result as "good."

CASE 9.—A man, aged 23, sustained a gunshot fracture of the left forearm which resulted in a loss of 3 cm. of the middle of the shaft of the radius and consequent nonunion. The soldier had received massage and physio-

Fig. 4 (Case 6).—Left humerus, five weeks after operation.

therapy, and the wound had been healed for three and a half months. A splint of tibial cortex was wedged into the medullary cavities of both fragments, and the extremity encased in plaster. Three weeks later when he passed from our observation alinement was good, and union was progressing satisfactorily. In a letter written to us more than five months after operation, the patient said that he had done well until the preceding week when he had fallen and broken the radius at the site of the new union.

CASE 10.—A man, aged 38, in a collision sustained a simple fracture of both bones of the left forearm at the junction of the middle and lower thirds. Five months later, there was malposition and faulty union and the fractured radius was reduced by open operation, holes being drilled in the fragments and kangaroo tendon being used to hold them in place. Four months later, as there was no union, a splint of tibial cortex was wedged into the lower frag-

Fig. 5 (Case 8).—Radius, six weeks after operation.

ment and laced in a cortical groove in the upper fragment. The ulnar fracture seemed to be the site of a fibrous union and was not disturbed. When he left our observation, nine weeks later, there was some backward bowing of the radius, but union seemed firm. Six months after operation, when last heard from, he wrote that "the upper end of the graft was loose but that union was progressing."

CASE 11.—A man, aged 31, sustained a gunshot fracture of the lower third of both bones of the left forearm. Débridement was at once performed, and six months later, a sequestrectomy. Two months after healing, during which time the patient received physiotherapy and vigorous massage, a tibial transplant was placed in the radius to bridge a defect of 6 cm. This tibial splint was wedged into the medullary canal of the upper fragment and laced with

Fig. 6 (Case 11).—Left forearm, six weeks after first operation.

kangaroo tendon in a cortical groove in the lower fragment. The ulnar fragments were joined by a fibrous union and were not disturbed. The patient did well, and one month later was transferred to another general hospital where his splint was inadvertently removed almost immediately after his arrival. When I next saw him, two weeks later, the splint had slipped out of the groove in the lower fragment and the condition was that illustrated by Figure 6. We performed a second operation, twelve weeks after the first,

when the upper end of the splint was so firmly incorporated with the proximal fragment that it was difficult to tell the one from the other. The lower end of the splint was brought back to its former bed in the distal fragment, but was fractured while being laced in position with kangaroo tendon. A second splint was then taken from the tibia and one end wedged in the distal fragment. The other end was laced to the original splint which had become incorporated with the proximal fragment. Three months later the roentgen

Fig. 7 (Case 11).—Left forearm, three months after second operation.

ray revealed some suggestion of union between the two transplants, and the patient had about 45 degrees of combined pronation and supination (Fig. 7). When last heard from, six months after the second operation, the patient reported that his forearm was "the same as before operation."

Case 12.—A man, aged 20, sustained multiple shrapnel wounds of the left leg and right arm and forearm. There was 5 cm. missing from the middle

third of the right radius with resulting nonunion. When he first came under our observation, the wound in the left leg was not quite healed. The forearm had been healed seven months and was held in pronation. Active supination was impossible, but about 50 per cent. of passive supination was present. The hand grip was weak. After massage and physiotherapy, a splint of tibial cortex was wedged into the medullary canal of the upper fragment and mortised in a groove in the cortex of the lower fragment where it was securely held by a lacing of kangaroo tendon. A cast was then applied from the middle of the humerus to the metacarpals, the elbow being flexed at a right angle. The wound almost immediately showed evidences of sepsis, and cultures revealed a streptococcus. It was concluded that the infection had been liberated from the scar tissue at the time of operation. When the patient passed from our observation, six weeks later, the wound was still septic and the upper end of the splint was loose.

Case 13.—A man, aged 28, sustained a gunshot fracture of the middle third of the left ulna which resulted in a loss of substance of 5 cm. and conse-

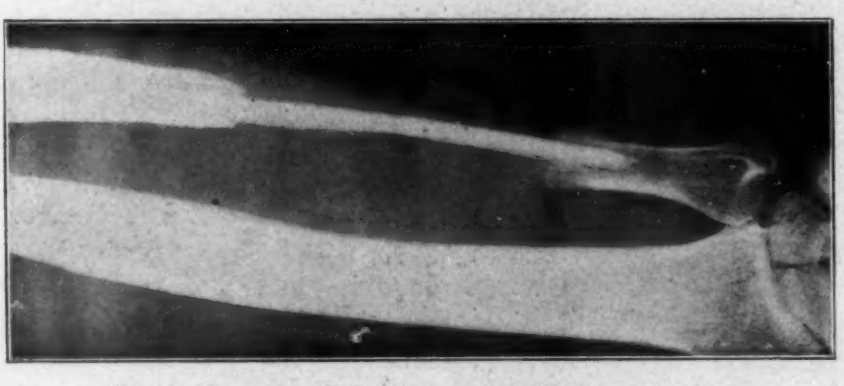

Fig. 8 (Case 13).—Left ulna, one month after operation.

quent nonunion. Three months after the wound had healed, during which time the patient received the usual preliminary massage and physiotherapy, a splint of tibial cortex was tightly wedged into the medullary canals of both fragments. The patient passed from our observation one month later, at which time the graft was in good position and the alinement was perfect (Fig. 8). When last heard from, five and a half months after operation, he said that there was good union at one end but that the other end was still ununited.

Case 14.—A man, aged 24, sustained a gunshot fracture of the middle third of the left ulna with considerable loss of substance. After the wound had been healed for thirteen months, during which time the patient had received several courses of massage and physiotherapy, the ulnar defect was bridged by a tibial transplant. A mortise was cut in both fragments, and the transplant was wedged in and tied in place by kangaroo tendon. The forearm was put up in moderate pronation. Six weeks later the union was apparently firm, but the cast was still strapped to the extremity (Fig. 9). When seen by one of us (W. H. T.), three months after the operation, the result seemed to be satisfactory in every way.

Case 15.—A man, aged 21, sustained a simple fracture of the right femur in a motorcycle accident. When he came under our observation, seven weeks after the injury, there was posterior angulation and an overriding of 15 cm.

After the overriding had been overcome by direct leverage with a piece of automobile spring, a splint of tibial cortex was wedged into the medullary canal of the upper fragment and laced with kangaroo tendon in a cortical groove in the lower fragment. The extremity was encased in plaster, the cast extending from the costal margin above to the toes below. Five weeks after operation, when the patient was transferred, the approximation was good and there was some callus present. When last heard from, nine months after operation, he was walking, and the result was all that could be desired.

CASE 16.—A man, aged 32, sustained a gunshot fracture of the middle third of the right fibula with a loss of substance of 4 cm. There was numbness over the dorsum of the foot, and dorsiflexion was impaired from weakness and involvement of the muscles in the peroneal scar and by contraction of the Achilles tendon. The wound had been healed four months, and the patient had received vigorous massage and physiotherapy. The anterior tibial

Fig. 9 (Case 14).—Left ulna, three months after operation.

nerve was freed from the scar, the Achilles tendon was divided and a tibial transplant was wedged between the cleared ends of the fibular fragments. The extremity was put up with the foot at right angles to the leg. Recovery was uneventful. When last heard from, nine months after operation, the patient said that his leg was as good as it had ever been.

CONCLUSIONS

1. It is possible for an intramedullary autogenous splint to become an integral part of the host's bone. This is shown by Case 11, in which we had an opportunity to observe the result of a previous bone graft operation. The proximal fragment and the upper end of the transplanted bone had grown together and appeared as one bone (Fig. 6). Transplanted bone is, then, not always absorbed.

2. In our cases the most frequent cause of failure was sepsis, the next in frequency was slipping of the internal bone splint.

CHRONIC DUODENAL DILATATION

ITS CONCOMITANT AND SEQUENTIAL PATHOLOGY

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Chronic duodenal dilatation is a frequent pathologic condition. This assertion is based, not alone on clinical observations, but also on the well known anatomic faults of the adult human duodenum. Normally the human duodenum has practically no mesentery; it is fixed dorsally by an abundant retroperitoneum, is indented in front and behind by blood vessels and is impinged on by contiguous structures. The quadruped has a long mesentery and freely movable duodenum. Comparative study shows that the human is paying the price of a potentially pathologic condition for the upright posture.

Embryologically, the human duodenum has a long mesentery and consequently free movement. Should the adult retain this condition the major etiologic factors of pathologic conditions of the duodenum would be removed.

The duodenum of the thirty to sixty day embryo is occluded by a vacuolated epithelial plug. Synchronous with the solid state of the gallbladder and cystic ducts, the first portion of the small bowel has a developmental condition not characteristic embryologically of any other portion of the small intestine. The vacuolated mucosa presents the potential etiology of duodenal diverticula found in the adult. The embryologic budding of contiguous structures into the dorsal and ventral mesogastrium draws the human duodenum partially into the position found in later life. However, part of the location in the adult is due to the upright posture and to Nature's effort to provide support for a structure subject to strain. The retroperitoneum fixes it, and as this shortens with the body's growth the last two thirds of the duodenum, through the medium of ligamentary developments and pancreatic growth, gradually assumes the adult position.

Histologically, the first 12 inches of the small bowel varies markedly from the rest of the lesser intestine. It contains all of the mucomembranous structures of the rest of the small bowel, namely: villi, glands of Lieberkühn, and mucous secreting cells, yet in addition to these, beneath its mucomuscular layer, are the glands of Brunner—a chain of isolated, racemose, bifurcating tubular glands. These secretory structures extend only slightly beyond the duodenojejunal angle, and are therefore rightly classified as strictly duodenal features. Comparative studies of these glands reveal a marked variation as to the

number present in different animal species; they are markedly scattered in the truly carnivorous type, fairly close together in the omnivorous and exist as almost a perfect sheet in the truly herbivorous, particularly the sheep. Such a comparative study would lead to the inference that the glands of Brunner are essential in car-

Fig. 1.—Normal relationship of first third of duodenum to pancreas and liver.

bohydrate digestion. When considering the etiology of glycosuria, further deductions from these facts lead one to believe that a diseased condition of the duodenum may favor the development of diabetes.

The first 4 inches of the duodenum has quite a different physiologic function to perform than have the second and third portions. Linked with the pylorus of the stomach at its beginning and having

the entrance of the common duct and the duct of Wirsung at its end, it receives a frankly acid chyme at its origin and an intensely alkaline secretion, derived from the liver and pancreas, at its termination.

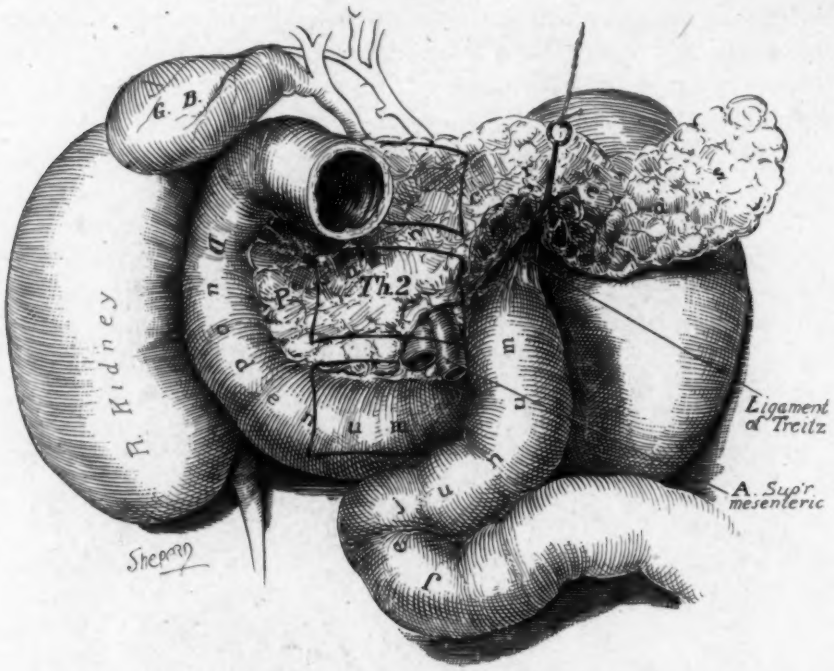

Fig. 2.—Normal C-shaped duodenum and the relationship of the superior mesenteric vessels to the last third of the duodenum, head of pancreas, gall-bladder and kidney.

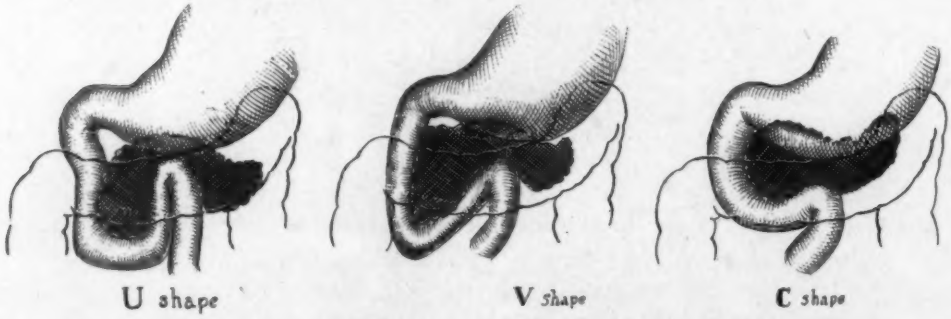

Fig. 3.—The three forms of duodenum: U and V, pathologic; C, normal.

This portion of the duodenum necessarily must serve as a transitional, anatomic, physiologic structure between the stomach and the small intestine proper.

The first 4 inches of the bowel have a complex function to perform, which is in no sense equaled by other parts of the duodenum, or the entirety of the small intestine.

Considerable clinical and physiologic evidence leads one to infer that the duodenum possesses racemose gland features with hormone qualities. Starling's isolation of prosecretin and secretin, the former changing by acid contact into the latter, sheds a light on the physiologic functions of the first portion of the duodenum. Considered at first as a probable enzyme, further tests, particularly its failure

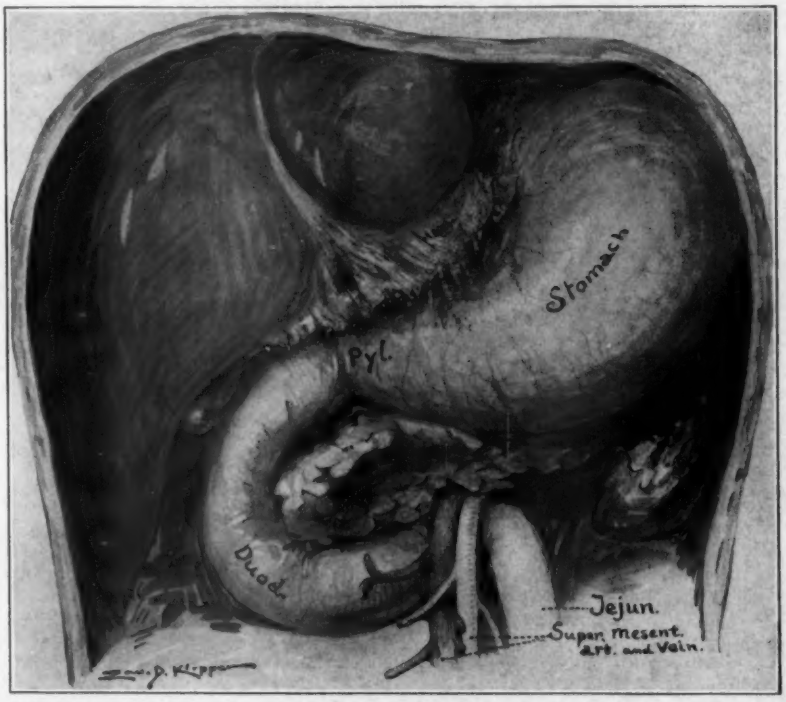

Fig. 4.—Normal anatomy of the stomach and duodenum and contiguous structures.

to be destroyed by heat, prove secretin to be, in all probability, a hormone. It has been demonstrated that this substance has several qualities: One stimulates the nerve mechanism of the pancreas with a consequent free flow of the pancreatic juice; another, a chemical entity, has intense acid neutralizing powers. Other secretin principles seemingly aid in gland interchange in establishing the normal when deficient, complicated pathologic conditions of the body may develop. The failure of the normal secretory functions of this portion of the duodenum would result in an improper digestive ability on the part

of the bile and the various enzymes of the pancreatic juice, due in the main to a deficient neutralization of the acid chyme with the necessary chemical change of the weaker alkalines by the more active acid stomach content. The sequence would be an amylaceous indigestion, a faulty fat transformation, failure of proteid change, a flatulent state, an intestinal indigestion, and, consequently, a poorly nourished body.

Maury,[1] in 1909, by a series of animal experiments, apparently showed that the duodenum, particularly the first third, was essential

Fig. 5.—Maury's experiments on animals.

to life. Despite careful transposing of the biliary and pancreatic ducts into a lower intestinal site, or connecting them with the stomach, duodenectomy resulted almost uniformly in death within seventy-two hours. The transposition of these ducts into a new location, after isolating them from the duodenum, did not produce death. The transfer of these ducts into a new location and the constriction of the duodenum lowered the vitality of the animal but did not kill it.

Matthews, in 1912, in the Lowe Laboratory, repeated these experiments with similar results and arrived at a similar conclusion. Moore-

1. Maury, J. W. D.: Intestinal Obstruction. An Outline for Treatment Based on the Cause of Death, Am. J. M. Sc. **137**: 725 (May) 1909.

head and Landes,[2] in the Lowe Laboratory, recently repeated these experiments on dogs and refuted Maury and Matthews' conclusions. Mann and Kawamura in the research department of the Mayo Clinic, in a large series of duodenectomies on various animals, arrived at conclusions similar to those of Moorehead and Landes. Despite the conclusions of the last four experimenters, clinical evidence sustains the belief that the duodenum is a complicated structure with vital functions. The rapid heart and anuria, subnormal temperature, obstipation and intense symptoms of shock associated with acute duode-

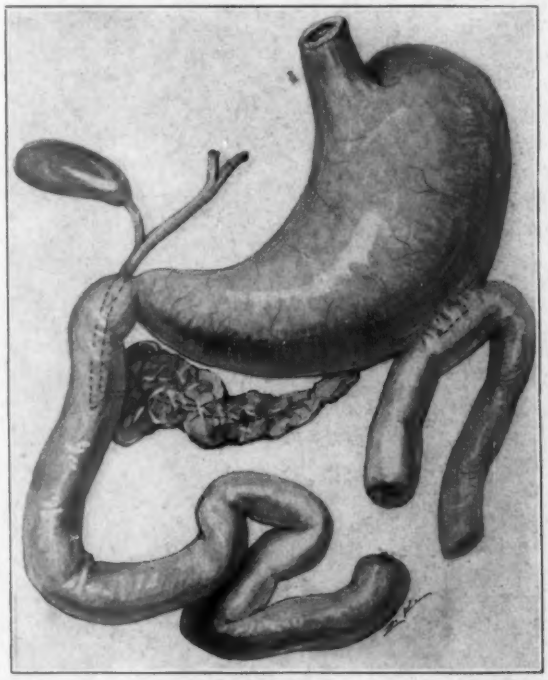

Fig. 6.—Experimental steps of Maury, 1909.

nogastric dilatation suggest that a secretion is formed by the duodenum which stimulates the vagus and lowers vasomotor tone.

The normal curve of the human duodenum simulates the curve of an Old English letter "C." Such a type rarely becomes dilated unless there exists a constrictive agent such as an embryologic band passing from the gastrohepatic omentum to the undersurface of the liver and gallbladder, or an adhesive band, of a developmental nature, from the transverse colon passing over the duodenum to the gallbladder and liver.

2. Morehead, J. J., and Landes, H. E.: Duodenectomy: a New Method, J. A. M. A. **72**:1127 (April) 1919.

The other two forms of the duodenum are the "V" and "U" type, each of which has pathologic possibilities. The former, on account of its low position, when accompanied by a gastrocolonic ptosis may give rise to symptoms which simulate those of chronic appendicitis. I have observed sixteen such cases, in each of which operation had been performed by others for chronic appendicitis with no relief to the patient.

Dwight,[3] of Harvard, has demonstrated beautifully by means of wax filling, the various forms and indentations of the human duode-

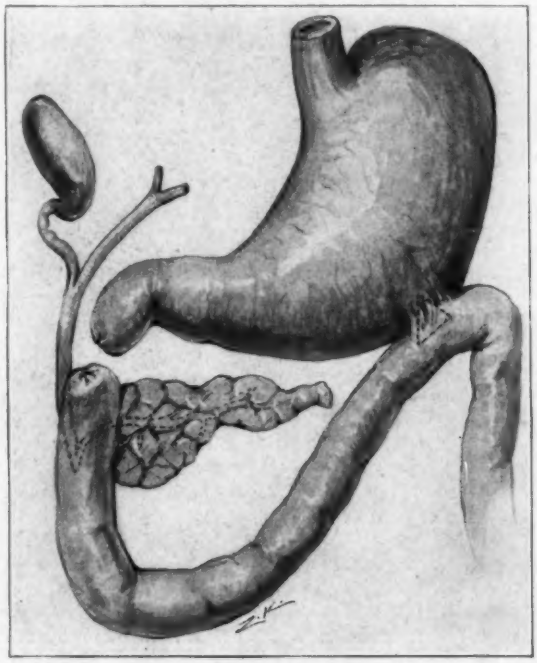

Fig. 7.—Experimental steps of Maury, 1909.

num and also the diverticula which may develop from it. His studies cover various stages from the infant to the adult.

Several fixation features of the adult type of the duodenum have potential points of pathology. Its first portion, which is partially covered by the peritoneum, normally has some movement; its inferior end is pressed on by the head of the pancreas and is covered by the transverse colon when the latter is in normal position. The beginning portion of the second part is also pressed on by the head of the

3. Dwight, Thomas: Observations of the Duodenum, J. Anat. & Physiol. **31**: 516, 1896.

pancreas, is almost entirely overlapped by the normally located transverse colon, and, at its juncture with the third portion, is indented behind by the second lumbar vertebra. The third portion is indented on its posterior surface by the inferior vena cava and abdominal aorta, while the superior mesenteric vessels compress it from the front. The last two thirds of the duodenum are fixed posteriorly by the retroperitoneum. The last third passes through a fold at the upper part of the mesentery where the retroperitoneum forms superiorly and inferiorly two folds, called the superior and inferior duodenal

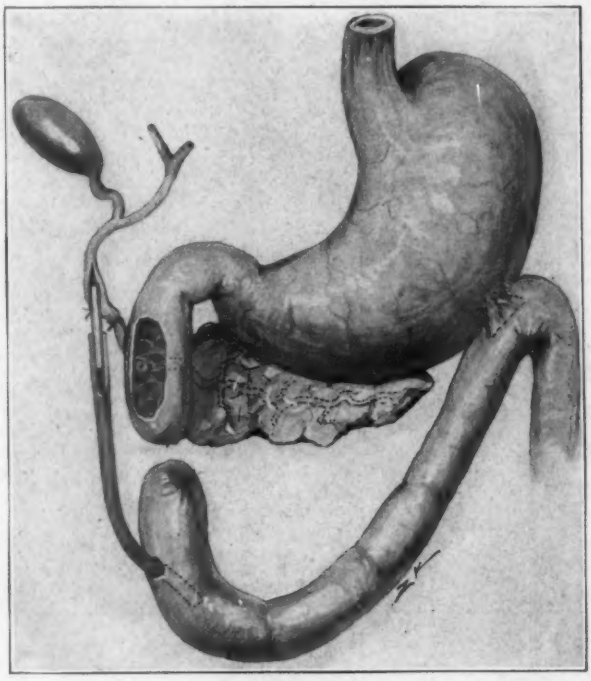

Fig. 8.—Experimental steps of Maury, 1909.

folds. The duodenum at this point enters into the arbitrarily named second portion of the small bowel, the jejunum. At this junction, on account of the retroperitoneal development of the ligament of Treitz, there exists a force indirectly fixing to the diaphragm the duodenojejunal angle. The ordinary sweep of the jejunum into the left renal fossa does not produce a marked but rather an obtuse angulation of the small bowel; should the jejunum drop directly down, an acute angulation occurs with a subsequent pathologic dilatation of the duodenum.

ETIOLOGY OF CHRONIC DUODENAL DILATATION

The etiology of chronic duodenal dilatation listed according to the frequency of each form is:

(*a*) Ptosis of the transverse colon, either alone or accompanied by gastric displacement, inducing through the mesenteric drag an approximation of the superior and inferior duodenal folds as well as a pull upon the ligament of Treitz.

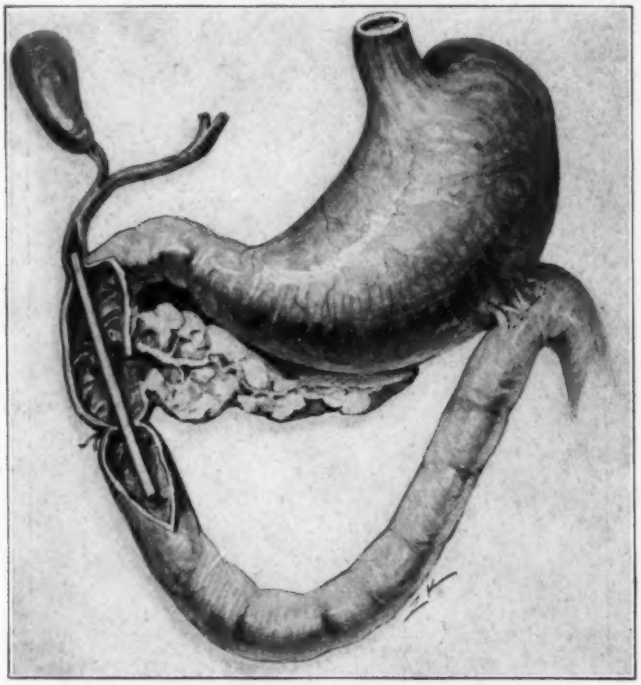

Fig. 9.—Experimental steps of Maury, 1909.

(*b*) A small bowel with short mesentery which prevents bony support in the pelvis for the intestines, producing duodenojejunal constriction similar to that of chronic ptosis; yet in addition to this, there is a pull on the superior mesenteric vessels which causes constriction of the last third of the duodenum.

(*c*) A jejunum dropping perpendicularly instead of having the normal sweep of the bowel into the left renal fossa; such direct drop produces a sharp angulation of the duodenojejunal angle.

(*d*) Embryologic remnants of the gastrohepatic omentum that pass from the pylorus to the gallbladder and undersurface of the liver with a constriction of the first third of the duodenum as a result.

(*e*) Developmental bands of adhesions from the transverse colon to the gallbladder, liver or pylorus, partially constricting the duodenum; the constriction of the adhesion in front acting with the vertebra behind as a resisting point.

(*f*) Diverticula of the duodenum and other obstructing factors are not uncommon. Many roentgenograms interpreted as revealing duodenal ulcer represent only this pathologic intestinal eversion.

(*g*) Troubles at the head of the pancreas, such as cysts, hematomas,

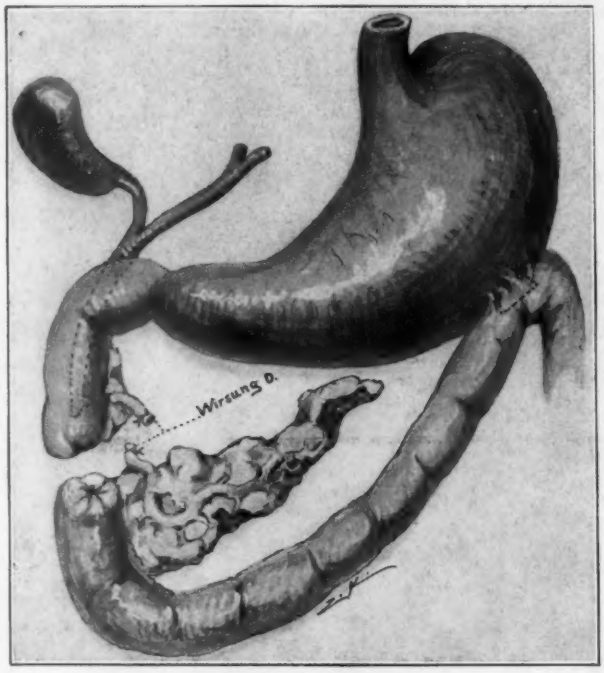

Fig. 10.—Experimental steps of Maury, 1909.

malignant growths and interstitial changes of the gland which, as it enlarges, impinges on and compresses the first and second portions of the duodenum.

(*h*) Exaggerated stimulation of the nerve supply of this portion of the intestine at times occurs as the result of pathologic conditions lower down. Such a nerve stimulation induces a clonic bandlike action on the part of the circular muscular fibers of the duodenum and if long continued produces duodenal dilatation. Ochsner's [4] duo-

4. Ochsner, A. J.: Constriction of the Duodenum Below the Common Duct, and Its Relation to Disease, Ann. Surg. **43**:80 (Jan.) 1906; Constriction of the Duodenum Below the Common Duct, and Its Relation to Disease, Tr. Am. Surg. A. **23**, 1905.

denal bands have been demonstrated by Boothby [5] to be simply thickenings of the muscular coat. Such bands have been proved to occur only occasionally in the second and third portions of the duodenum.

SYMPTOMATOLOGY

The symptomatology of chronic duodenal dilatation is marked by distress in the right half of the epigastric region, at first occurring nearly always from three to four hours after a meal. Ultimately this symptom becomes constant, varying only in its degree of severity.

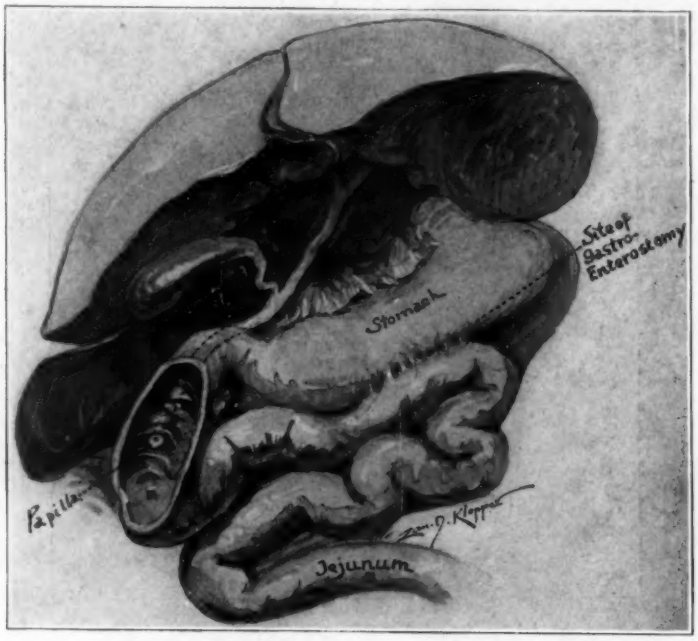

Fig. 11.—Experimental steps of Maury, 1909.

There are belching, marked autointoxication, accompanied by constipation, an irritable, rapid irregular heart, which cannot be explained by a cardiac disturbance or by exophthalmic goiter, neurasthenia, headache of a hemicranial type and scanty urine of high specific gravity, not always laden with indican.

The diagnosis of chronic duodenal dilatation is based on the clinical history with these symptoms plus careful roentgen-ray examinations. Roentgenograms should be taken serially, ten to twenty

5. Boothby, W. M.: The So-Called "Ochsner Muscle" of the Duodenum, Boston M. & S. J. **157**:80 (July 18) 1907.

minutes apart, for an hour, and then a six-hour roentgenogram taken in a prone, lateral posture, as for motor inadequacy of the stomach. Hayes' deep percussion test over the right rectus muscle in the area bounded by the liver to the right, the normal transverse colon line below, and the pylorus to the left, gives an unnatural, gaslike note

Fig. 12.—Experimental steps of Maury, 1909.

when duodenal dilatation exists. The paradoxical pressure test of Hayes [6] is made practically as Quimby's [7] drag, only the diagnostician makes the pressure pull to relieve duodenojejunal constriction, thus allowing the duodenal gas content to pass into the jejunum while

6. Hayes, W. V. V.: Chronic Intestinal Stasis, New York M. J. **119**:170 (Jan. 24) 1914.

7. Quimby, A. J.: Chronic Intestinal Stasis, New York M. J. **119**:162 (Jan. 24) 1914.

listening, with the stethoscope, for the phenomena at the normal point of junction of these two portions of the small bowel.

Testing for starch in the stools and Einhorn[8] duodenal content tests complete the methods that I use to differentiate other right hypogastric troubles from chronic duodenal dilatation. The concomitant pathology is dilatation or atony of the stomach which is only too frequently classified as an isolated instead of an accompanying trouble. The sequential pathology is extensive. The reversal of the flow of duodenal contents from intestine to stomach leads to marked digestive disturbance. The dilatation of the duodenum obstructs the normal output of the glands of Brunner, which results in a faulty

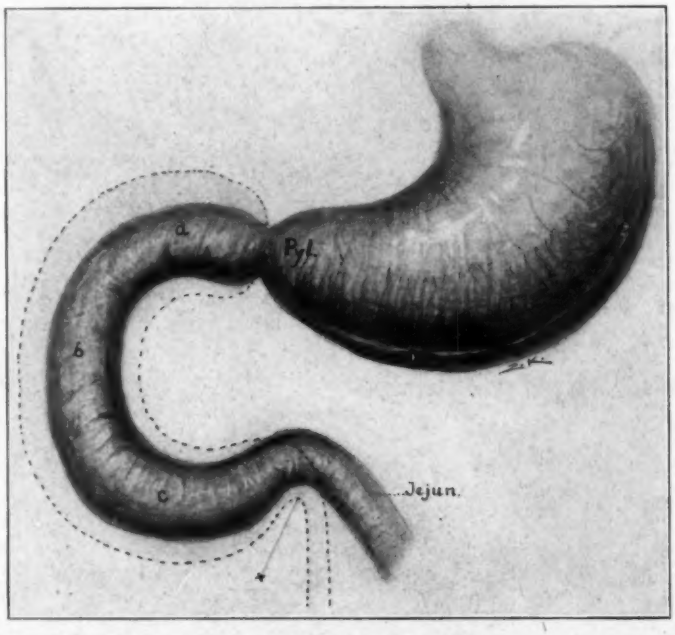

Fig. 13.—Normal sweep of jejunum into left renal fossa. Dotted line shows results of drop of jejunum with consequential dilatation of the duodenum.

secretin development with a sequential lack of alkaline neutralization; hence, amylaceous indigestion, flatulency, toxicosis, cardiac, nerve, ocular, cerebral and renal troubles, hemicrania, exophthalmic goiter and at times idiopathic epilepsy are found. A history with a symptomatology covering these major points, particularly a story of chronic gastric trouble and intestinal stasis, should lead the diagnostician to investigate the duodenum.

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8. Einhorn, Max: Historical Sketch of the Development of the Duodenal Tube, Am. J. M. Sc. **151**:202 (Feb.) 1916.

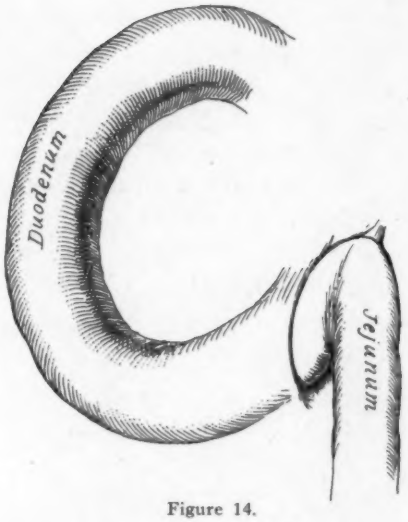

Figure 14.

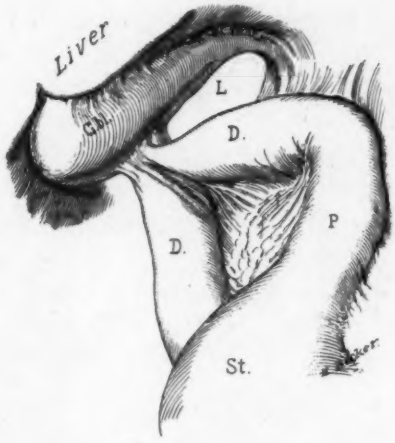

Figure 15.

Fig. 14.—Duodenojejunal angle: superior and inferior folds, with direct dropping jejunum.

Fig. 15.—Angulations of the duodenum, produced by an old duodenal ulcer, attached by adhesions to the gallbladder.

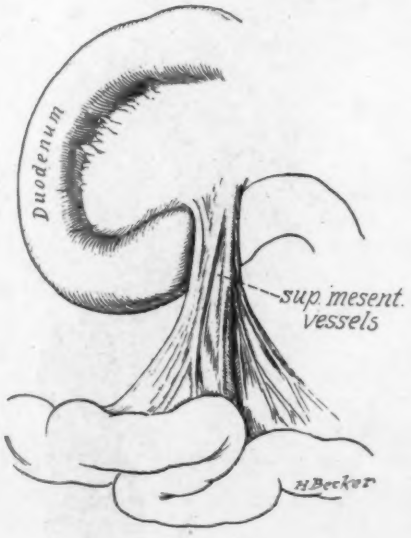

Figure 16.

Fig. 16.—Short mesentery: constriction of the duodenum by superior mesenteric vessels.

The treatment of chronic duodenal dilatation is medical, mechanical and surgical. The positive correction of this trouble is a purely surgical procedure. I have been able, however, to give a large per-

Figure 17.

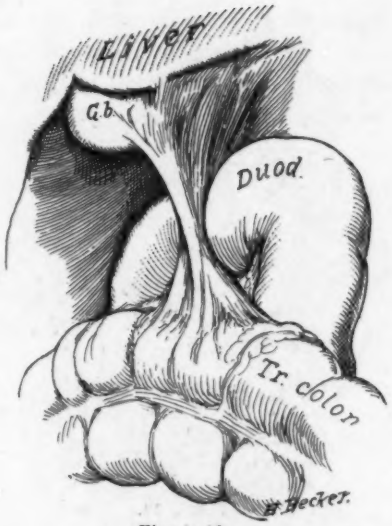

Figure 18.

Fig. 17.—Developmental band from gastrohepatic omentum, passing from the pyloric end of stomach to gallbladder, constriction of first third of duodenum.

Fig. 18.—Developmental band following ulceration of transverse colon passing over duodenum to gallbladder, causing constriction.

Figure 19.

Fig. 19.—Embryologic band from gastrohepatic and greater omentum, showing the blood vessels existing in an embryologic structure instead of absence of blood supply as is the case in developmental bands, which produces duodenal stasis and dilatation.

centage of my patients marked relief by the following means: first, rest, and rest in a recumbent posture, demanding that the patient, two hours after each meal, assume the knee-hand position for from ten to fifteen minutes, in order that the mesenteric pull may be removed. Then

Fig. 20.—Gastroptosis, anterior view, showing angulation of duodenum, ptosis of transverse intestine with consequential drag on right kidney.

the patient turns to the right side and, with the hips elevated, the passage of the duodenal content to the jejunum is stimulated instead of being emptied reversely into the stomach or remaining in the obstructed bowel. The patient is given a carefully selected composite

nitrogenous, glucose charged, carbohydrate diet. Foods are selected for their low ferment and high fat producing qualities. Broiled steaks, mutton chops, chicken, breakfast bacon, soft boiled and poached eggs, roast beef, medium well done, roast mutton and chicken, stale or

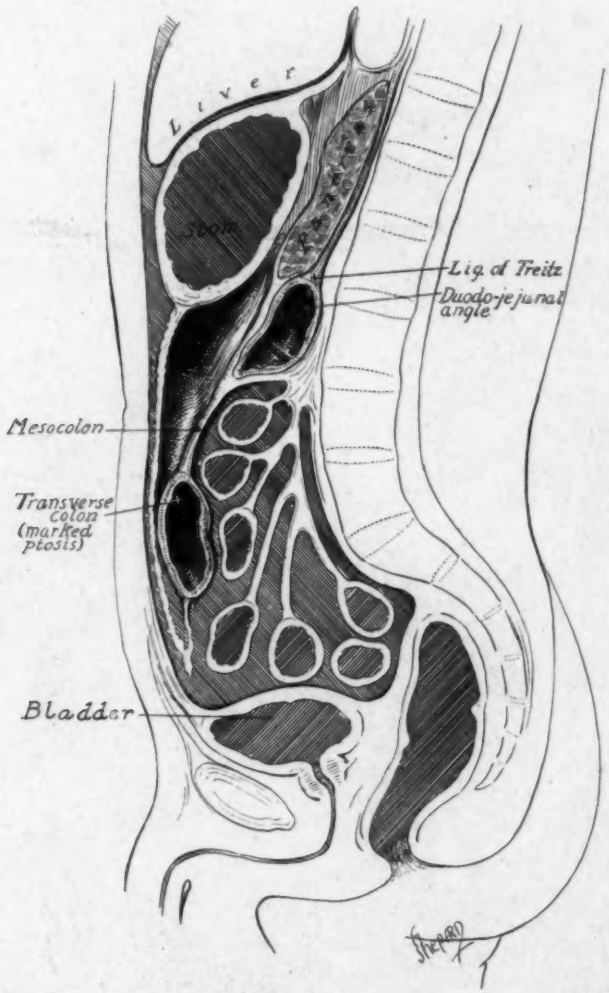

Fig. 21.—Transverse colon ptosis with constriction by ligament of Treitz, Bloodgood pathologic condition.

toasted white bread, baked or boiled Irish potatoes, butter in abundance, lettuce, spinach, toasted cereals, rice (Chinese style), sweet milk and cream, if there does not exist an idiosyncracy to them, are recommended. After the completion of each meal, a raw, salted egg, covered with orange juice is taken.

To commence with, if possible, a pure milk diet is given, and the change is gradually made to the diet mentioned while the patient is still in bed. When the patient is permitted to be up, a mechanical support made of ducking is fixed inside a front-laced corset and applied before

Fig. 22.—Billroth's duodenojejunostomy; improper technic.

the patient arises and while in the heel-shoulder position; in the meantime the stomach is dragged up, as a woman with a pendulous abdomen confines her too abundant abdomen while lacing a corset. In fact, the same method is used as when dealing with the gastrocolonic ptosis as far as the support goes.

Before food, the following prescription, or a variation of it, is given:

℞ Tinct. belladonnae foliorum..... ♏ iii
Tinct. nucis vomicae..... ♏ v
Tinct. cardamomi compositae ..... ♏ xx
Aquae destillatae.....q. s. ad fl. ℥ i

Fig. 23.—Bloodgood-Crouse suggestion: second third of duodenum and jejunum approximated.

After the ingestion of food this prescription is given:

℞ Magnesii oxidi ..... ℥ iv
Sodii bicarbonatis..... ℥ iiss
Olei menthae piperitae.....q. s. ad fl. ℥ iv
M. Sig.: One teaspoonful in half a glass of water.

The former is given to check the excessive secretions and relieve spasms, and the latter to neutralize excessive acidity.

During the entire course of the treatment the duodenal Einhorn lavage is utilized, consisting of 16 ounces of a 3 per cent. hypertonic

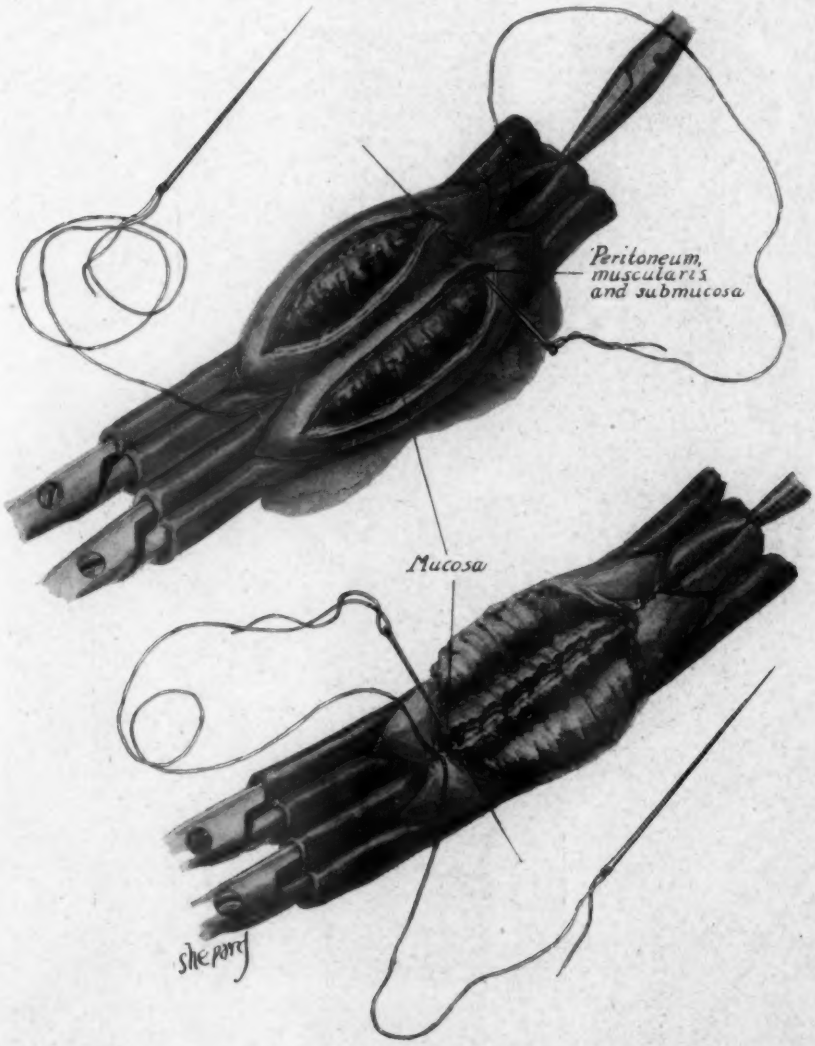

Fig. 24.—Methods of approximation.

salt solution, which contains 2 drams of sodium sulphate. This is done each morning and is easily accomplished provided the patient is encouraged to sip a half to one teacupful of warm water while the tube is still in the stomach, to encourage the passage of the tube

into the intestine. The tube is lowered until the yellow, golden-colored, duodenal content appears, and subsequent to such appearance the lavage is started; this should occupy about twenty minutes. This should be done daily for about a week, then on alternate days and thus gradually the intervals should be lengthened, but the procedure should

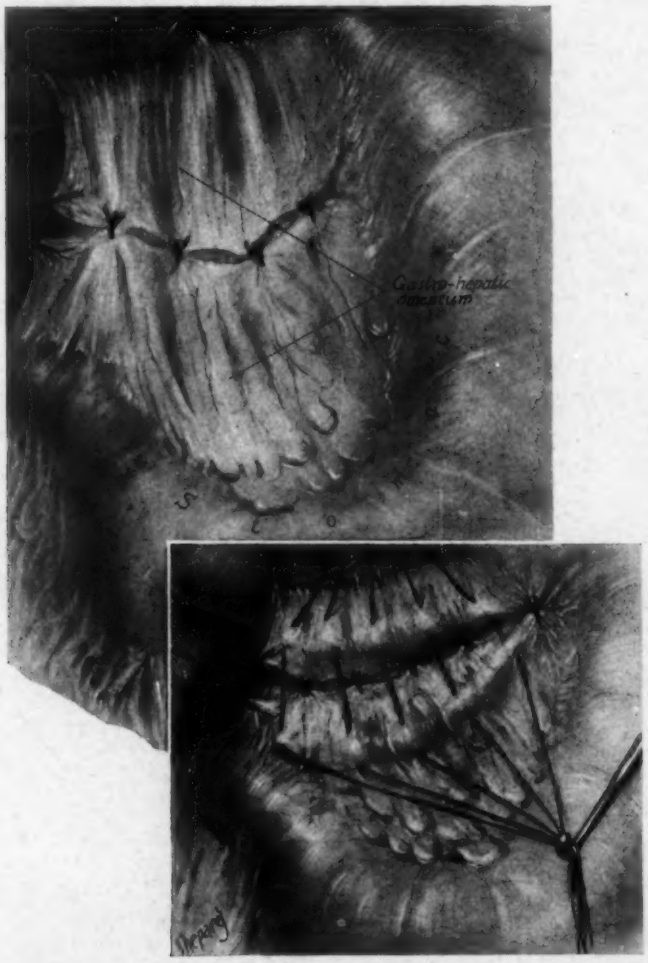

Fig. 25.—Bayes operation for shortening the gastrohepatic omentum.

be repeated occasionally for months. As a rule within two or three hours after the lavage, two or three copious watery stools occur resulting in a feeling of well-being.

While one can expect alleviation through these methods in the ordinary case, the real curative treatment is strictly surgical. Surgical intervention should, of course, consist in removing

the causes whether they be embryologic or pathologic bands, ptotic colon alone or stomach displacements as well. In gastrocolonic misplacement the Bayes operation: plication of the gastrohepatic omentum, amplified by my reinforcement of it by utilizing the so-called falciform ligament, is the method of choice. The falciform and round ligaments are detached from their umbilical and abdominal wall attachments, leaving the diaphragm and liver fixation points alone. The falciform ligament, which is but an embryologic winglike fold of the abdominal peritoneum, is split and sutured with the raw

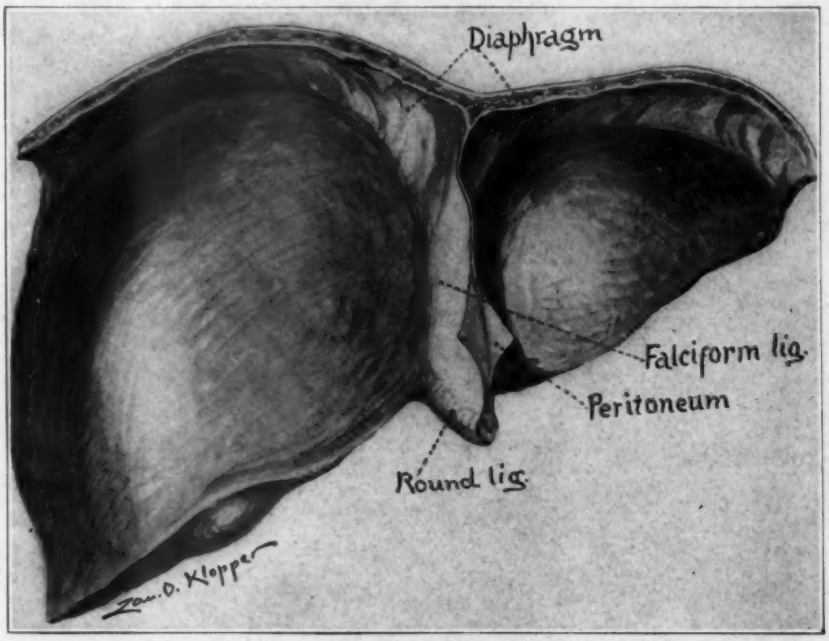

Fig. 26.—Falciform ligament, showing relationship of the falciform ligament to diaphragm and liver.

surface downward, together with the round ligament, over the plicated gastrohepatic omentum with No. 1, twenty-day chromic catgut. Adhesions should be dealt with according to their nature.

A cholecystectomy is indicated when a gallbladder is markedly thickened and adherent. I am still at a loss to know how to deal with a raw-surfaced gallbladder, except by its removal, thus avoiding the reforming of adhesions. The Bloodgood [9] method of performing a par-

9. Bloodgood, J. C.: Dilatation of the Duodenum, J. A. M. A. **59**:117 (July 13) 1912; Acute Dilatation of the Stomach. Gastro-Mesenteric Ileus, Ann. Surg. **46**:736, 1907.

tial colectomy in duodenal dilatation caused by ptotic colon has, in his hands, led to successful results. A developmental or congenitally dilated colon should be removed when co-existing with the duodenal dilatation.

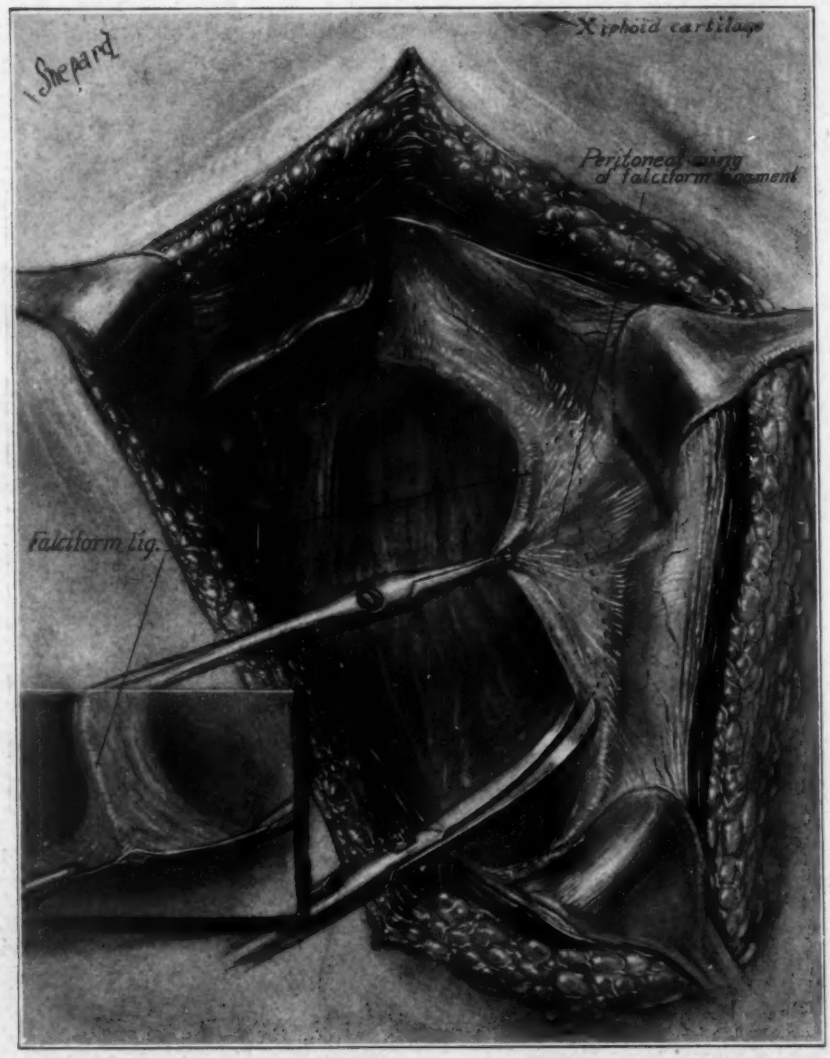

Fig. 27.—Crouse technic: utilizing the falciform ligament and its peritoneal wing. Inset shows method of stretching before splitting.

The logical course to pursue in all cases of widely dilated duodenum, as suggested by Bloodgood and followed by Stively in 1910, is to perform a duodenojejunostomy of the lateral apposed type (not the Billroth utilization of the first third of the duodenum, with its

consequential need of tearing through the gastrocolic omentum in order to approximate the jejunum to the duodenum), an approximation of the second third of the duodenum with the jejunum under the temporarily elevated transverse colon. A direct drain of the important

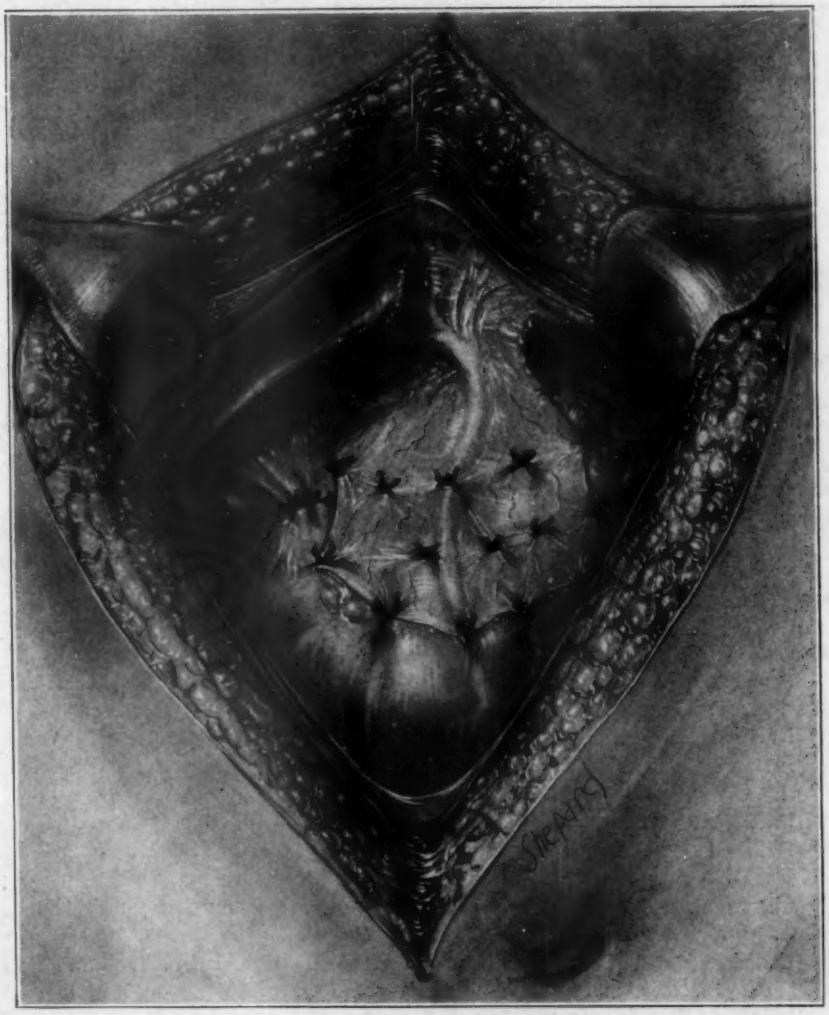

Fig. 28.—Crouse technic: use of detached falciform and round ligaments as an addition to the Bayes operation.

first part of the duodenum occurs and a shoe-fly avoidance of the obstructed third part results with a consequential rapid subsidence of duodenal toxicosis. A diseased appendix found at operation should be removed.

I shall not give case histories to exemplify the points brought out in this article, for I believe that a composite picture of many different cases of duodenal obstruction is far more elucidating and less time consuming than the citation of individual case reports.

My experience in forty-nine cases has been medical in the main, as only five patients consented to some form of surgical operation.

CONCLUSIONS

There is, in all probability, a chronic, pre-existing duodenal dilatation in all cases of acute gastroduodenal dilatation. The duodenum should be inspected or at least palpated the same as the gallbladder or the appendix, in all abdominal operations where such palpation does not disseminate infection.

Gallbladder, gastric and colonic operations are not complete without the inspection of the first third of the duodenum, the lifting up of the transverse colon and investigation of the retrocolonically placed duodenum as well as the duodenojejunal fold.

Duodenal dilatation is a frequent and not a rare condition.

It is cured by surgical intervention.

Duodenojejunostomy will cure most cases of duodenal ulcer.

Many patients with gastric ulcer treated by posterior, no-loop, gastro-enterostomy technic, should have a duodenojejunostomy performed as well.

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A ROENTGENOLOGIC STUDY OF THE COURSE OF POSTINFLUENZAL PYOPNEUMOTHORAX*

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During the last few months we have had the opportunity of studying roentgenologically the course of four cases of postinfluenzal pyopneumothorax, and we believe that the pathologic pictures obtained during these studies are worthy of report.

These patients have all been treated as conservatively as possible and in only one case was recourse had to a radical surgical procedure.

All roentgenograms were stereoscopic. They were made with a hydrogen tube, with a 33-inch target-plate distance. The patients were in the erect position and both exposures were made during one inspiration.

REPORT OF CASES

CASE 1 (No. 1948).—*History.*—A man, J. S., aged 18, entered the hospital Jan. 27, 1919, complaining of severe, stabbing pain in the right chest. Two years before, he had had an attack of pleurisy which lasted five days. In November, 1918, he had sharp, stabbing, inspiratory pain in the lower right chest, and for three weeks following, a dry unproductive cough. December 25, he was tapped by his home physician and a quart of fluid, said to be bright red, was removed. One week later he was tapped with a similar result and again on January 6, 1 or 2 ounces was aspirated (Fig. 1).

Examination.—February 15, he was tapped and thick purulent material was obtained. The right chest was found to be practically immobile and in front from the level of the third rib down, percussion elicited dulness to flatness with diminished to absent breath sounds. The left chest was hyperresonant with exaggerated vesicular breathing. The apex of the heart was found displaced 2 finger breadths to the left of the midclavicular line. Blood count revealed: red blood corpuscles, 3,860,000; white blood corpuscles, 14,900. Differential count showed 80 per cent. polymorphonuclears. The urine was negative. The sputum was repeatedly negative for tubercle bacilli.

Clinical Course.—During January and February, the daily temperature ranged between 100 and 101.5 F. During March (Fig. 2) it became normal and remained so until April 18, when he was discharged (Fig. 3). He was readmitted April 24. The physical examination was essentially the same as

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at the time of his original admission except that the upper level of dulness was now at the fifth rib. The clinical course was uneventful; the temperature was normal on admission and remained so; the color was good and he gained in weight steadily until June 4. When he was discharged, his weight was equal to his previous best weight. At this time only slight dulness remained at the right base, and his general condition was excellent (Fig. 4).

Fig. 1 (Case 1).—Condition Jan. 29, 1919: curious bands of thickened pleura, some air-containing loculi, presenting an appearance not unlike that of a diaphragmatic hernia of a gas-filled bowel.

Jan. 7, 1920, he returned for reexamination (Fig. 5). His weight remained unchanged, but he complained of rather poor appetite. Examination was entirely negative.

July 20, he again returned for examination (Fig. 6), stating that in February, he had had double pneumonia and had been in a hospital for six weeks. Since that time he had had some pain in his right chest. Six weeks before a tonsillectomy had been performed and three weeks later the submucous

membrane had been resected. His general appearance was excellent. His weight was normal. Percussion revealed slight impairment of breath sounds over the right apex in back, but the examination was negative otherwise.

Roentgen-Ray Findings.—Jan. 29, 1919, a roentgenogram revealed asymmetry of the thorax with emphysema on the left side. The apex of the heart was above the diaphragm in apparently normal position although the upper portion of the heart shadow and the shadow of the superior mediastinum, including the trachea, were distinctly displaced to the left, that is, away from the side in which the pathologic changes were most marked. On the right, there was

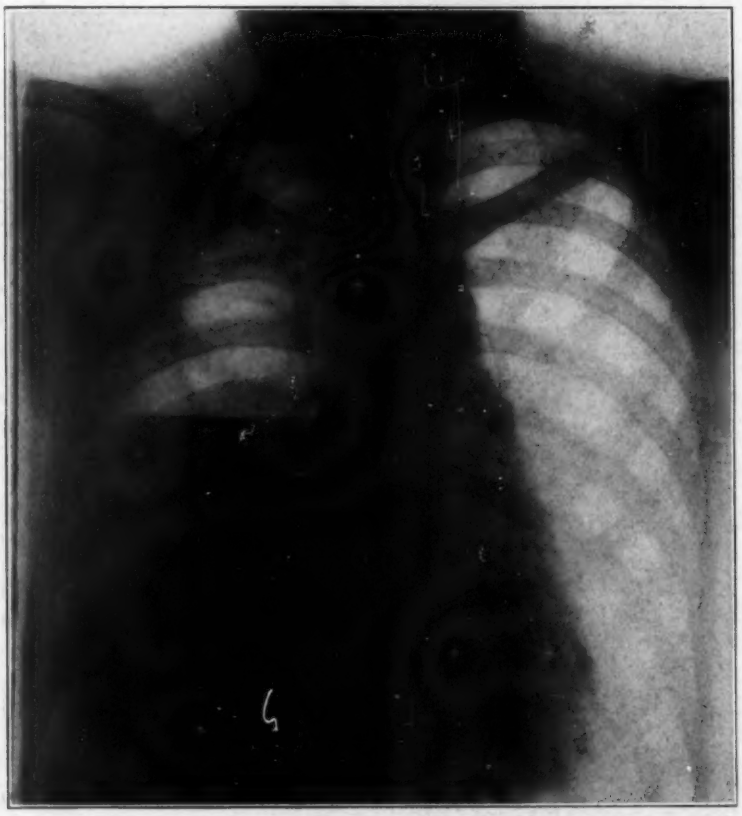

Fig. 2 (Case 1).—Condition March 3, 1919: typical straight line marking upper border of air and fluid in the pleural cavity. The pleural bands shown in Figure 1 can still be seen above the level of the fluid.

a series of air bubbles which on superficial examination suggested the presence of the large bowel in the pleural cavity. Closer examination, however, revealed the fact that there was not a complete continuity of the different cavities. The lower shadow was comparatively high, and the parietal pleura was enormously thickened. It was probably half an inch thick. There was narrowing of the interspaces, and what we believed to be the retracted lung had pathologic density which was fairly uniform and of markedly altered texture. The alternative interpretation assumed the increased density over

the liver shadow to be due to pleural exudate and the density of the lung to be due to a similar cause. Examination after ingestion of barium disclosed the fact that the shadows in the pleural cavity were not connected with the alimentary canal.

Feb. 16, 1919: As compared to the preceding findings, there was a reduction in the thickness of the parietal opacity and of the shadow adjoining the posterior mediastinum. The broad bands noted previously subdividing what seemed to be the cavity of the thorax were reduced in width. The cavity was

Fig. 3 (Case 1).—Condition April 18, 1919, showing the greatest extent of the effusion.

more nearly continuous, and we could definitely recognize some sort of bronchovascular tree in this space. The opacity in the lower part was not quite so extensive, but was still of high degree and rather poorly defined above.

March 3, 1919: These roentgenograms revealed a horizontal fluid level in the midthorax and in the region that was clear formerly. Obviously the chest now contained both air and pus beneath a very dense, thick pleura.

April 18, 1919: The entire right side was relatively more dense than before. The fluid level could still be made out high up beneath the second rib, above which was a small air bubble.

June 27, 1919: Some retraction of the fourth and fifth interspace was noted on the right. There was pleural thickening in both apexes, particularly marked on the right and continuing practically to the diaphragm on this side. The pleural angle was obliterated on the right by a definitely demarcated area of great density, but there was an area of comparatively normal lung, mesial and inferior to the part in which the pathologic changes were noted. There was no sign of definite fluid level or air.

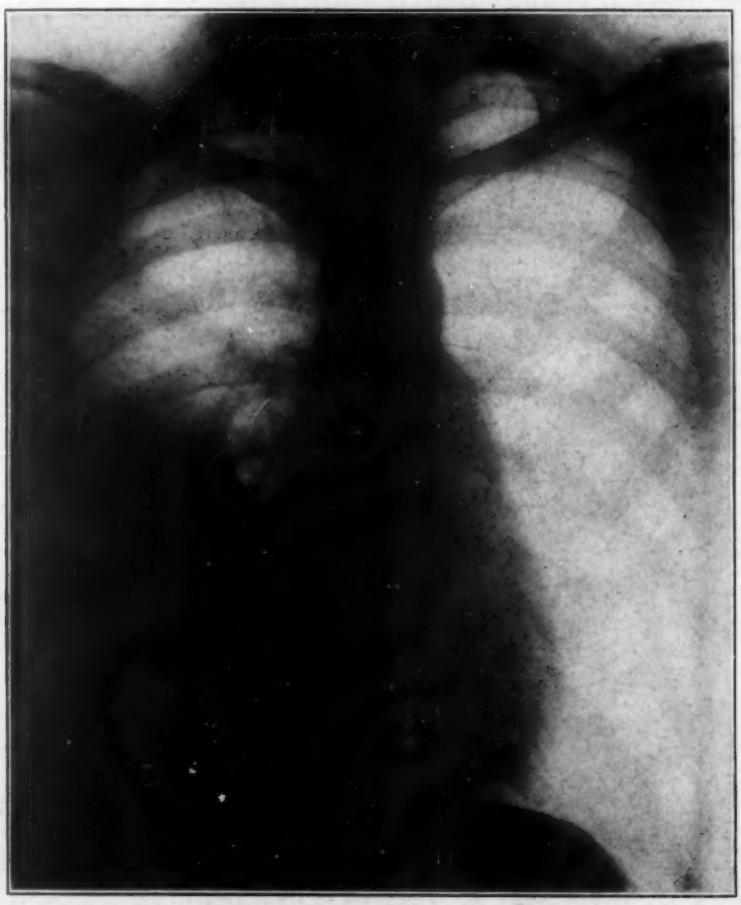

Fig. 4 (Case 1).—Condition June 27, 1919: beginning encapsulation and resorption of fluid and exudate. The appearance of lung markings may be noted in the upper half of the pleural cavity.

July 8, 1920: There was an insignificant flattening on the right side above and a slight pleural line throughout the length of the axilla. On the right side, the apex was slightly obscure as though from thickening of the pleura on the anterior and posterior surfaces not seen in cross-section. All this could be easily missed.

July 21, 1920: There was definite pleural thickening at the right apex and axilla. Otherwise the chest was entirely negative.

CASE 2 (No. 2010).—*History.*—P. D., a baggageman, white, aged 37, with a negative family and personal history, whose chief complaint was cough and weakness, entered the hospital, Jan. 10, 1920. The present illness began in December, 1918, with an attack of influenza which was epidemic at that time. The patient was in bed until February, 1919. Shortly after getting up he began to cough and raise considerable greenish, foul-smelling sputum, a teacupful a day. The amount of sputum had gradually decreased, although the

Fig. 5 (Case 1).—Condition Jan. 6, 1920: the only evidence of the preceding process is a fine pleural line in the axilla and slight deformity of the right diaphragm.

fetor continued. During the summer, he had paroxysms of coughing which lasted from five to ten minutes, during which he would become very faint and perspire freely.

Examination.—This revealed rather poor nutrition, exaggerated excursion of the right side, and fixation on the left. The left chest showed a flattening of an area about the size of the patient's palm below the clavicle. There was slight hyperresonance on the right, with vesicular breathing, unchanged

fremitus and no râles. Slight resonance at the uppermost part of the right apex was noted. Elsewhere there was flatness. Except at the apex, no breath sounds were heard: there they were distant and metallic in character, but unchanged in quantity. On shifting the patient to the prone position, the percussion note over the back changed from flatness to dulness. Clinical findings were: hemoglobin, 85 per cent.; red blood corpuscles, 4,300,000; white blood corpuscles, 13,000. The Wassermann reaction was negative. The urine was negative. The blood pressure was 120/90. The sputum was negative for tuberculosis.

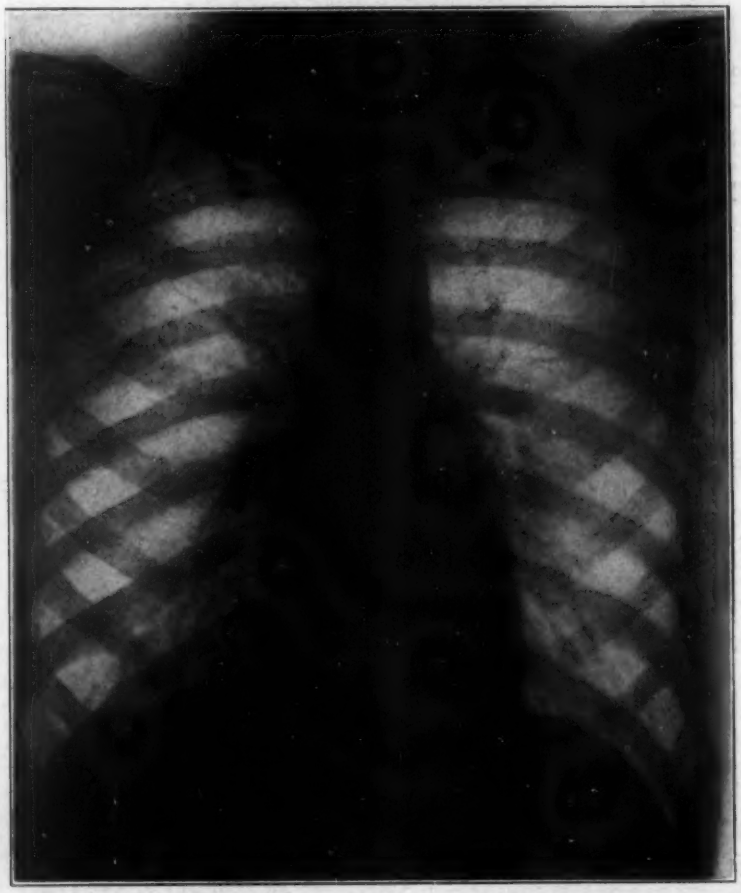

Fig. 6 (Case 1).—Condition July 21, 1920, after a second attack of influenza and double pneumonia, following which the patient underwent a tonsillectomy and a submucous resection. Practically no residue may be noted.

Clinical Course.—The temperature fluctuated between 97 and 99 F., on one occasion reaching 100 F. (Figs. 7 and 8). The patient was aspirated, January 14. Cultures after diagnostic puncture demonstrated no growth. January 16, 600 c.c. was aspirated; January 29, 450 c.c.; February 16, 1,000 c.c.; February 25, 700 c.c., which showed white cells, 33,000, and March 9, 750 c.c., which

showed white cells, 30,000. White cell count rose so that on March 9 he was transferred to the surgical division for thoracotomy. Smears from the fluid removed, all showed streptococci, but no growth was ever obtained.

March 11, blood examination revealed: hemoglobin, 85 per cent.; red blood corpuscles, 4,800,000; white blood corpuscles, 16,000. Urine examination was negative. March 13 a portion of the ninth rib was resected, drainage was instituted under local anesthesia and the patient made an uneventful recovery.

Fig. 7 (Case 2).—Condition Feb. 5, 1920, before aspiration.

He was discharged March 22. The temperature was normal (Fig. 9). July 19, the patient had gained 15 pounds and was gaining strength. He felt perfectly well (Fig. 10).

Roentgen-Ray Findings.—Jan. 10, 1920, these were pathognomonic of a fluid exudate in the left chest modified by a gas bubble. The fluid exudate was separated from the lateral chest wall by an exudate one-half inch in thickness. Enormous compensatory emphysema was noted on the right side.

There was no displacement of the heart or mediastinum, in fact the cardiac shadow was almost entirely overshadowed and might actually have been retracted to the left.

Jan. 24, 1920: After aspiration, considerable density was noted in the left lower chest, which was entirely pleural in character. This was found chiefly from the fifth to the eighth interspaces, and on the lateral border covering the entire left side from the first to the ninth rib. There was some air on the left, along the lateral border outside the lung from the fifth to the eighth

Fig. 8 (Case 2).—Condition Feb. 5, 1920, after aspiration: expansion of lung and thickness of pleura in the axilla may be noted.

interspaces. The bronchovascular tree on this side was rather prominent. There was no longer any definite emphysema on the right side, and the heart was normal in size, shape and position.

Jan. 29, 1920: After aspiration, on the left the diaphragm was still higher than normal and on this side there was a small amount of air encapsulated in the pleura though considerably less than at the previous examination. The greater part of the parenchyma of the left lung contained air; distinctly more than when the previous examination was made.

Feb. 6, 1920: When compared to the previous roentgenograms the lung showed considerable more expansion and the thickness of the pleural exudate was considerably diminished. There was more retraction of the interspaces and the heart was displaced rather more to the right.

Fig. 9 (Case 2).—Condition April 29, 1920, after operation, showing drainage tube in place. Retraction of entire left chest and thickness of pleura throughout the entire length of the axilla may be noted. Lung markings may be seen everywhere except in the extreme pleural cavity.

Feb. 16, 1920: Eleven days after aspiration, two sets of roentgenograms were made before and after aspiration. The former showed a horizontal fluid level at the level of the second rib in front, with a gas bubble, apparently mostly in front. There was a small circumscribed annular shadow in the extreme apex outside a rather prominent line running down the pleura in front which was evidently an encapsulated pocket.

A stereoscopic roentgenogram taken after aspiration showed the lung partially but imperfectly expanded. The left lower lobe was distinctly thicker and denser than the one on the opposite side. The lower margin could be well made out. A curvilinear shadow along its lower border outlined a gas bubble, which, contrary to expectation, did not lie in the upper portion of the thorax, but immediately above the diaphragm. Marked thickening of both the visceral and parietal pleurae was noted in the middle third of the chest. The encapsulated annular shadow in the apex remained as before. Narrowing of the thorax on this side and displacement of the trachea persisted. As

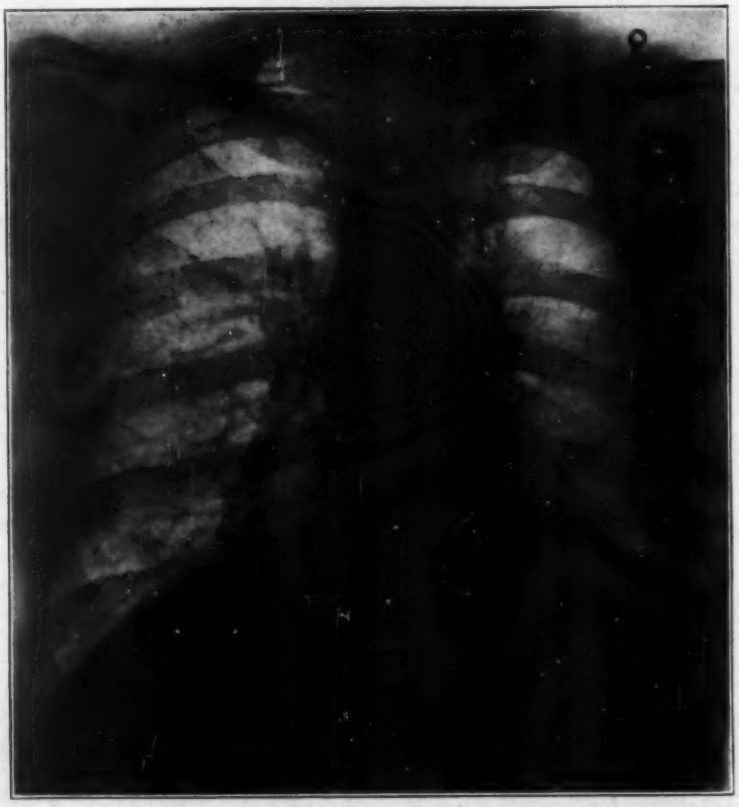

Fig. 10 (Case 2).—Condition July 20, 1920: although there is marked contraction of the left chest, the lung on the affected side is functioning. The patient has regained his normal weight.

a whole, the lung, which was now expanded, was denser than the one on the opposite side. Evidently these differences in density were due to blood.

Feb. 25, 1920: The roentgenograms were practically identical with the preceding.

March 10, 1920: The line representing the thickened interlobar pleura was not at all marked in these roentgenograms as compared to the previous ones. The lung was much better distended and extended laterally to within an inch and a half of the pleural margin.

April 30, 1920: After thoracotomy, roentgenograms showed adequate drainage. The pleural thickening persisted.

May 28, 1920: The lung itself was less dense than previously. There was marked retraction of the entire left chest. Pleural thickening was seen laterally from the apex to the base, but less marked than before.

June 22, 1920: The lung was well expanded but the broncho-vascular tree was especially clear. The pleura was definitely less dense, and the pleural angle was very much more free than at the previous examination.

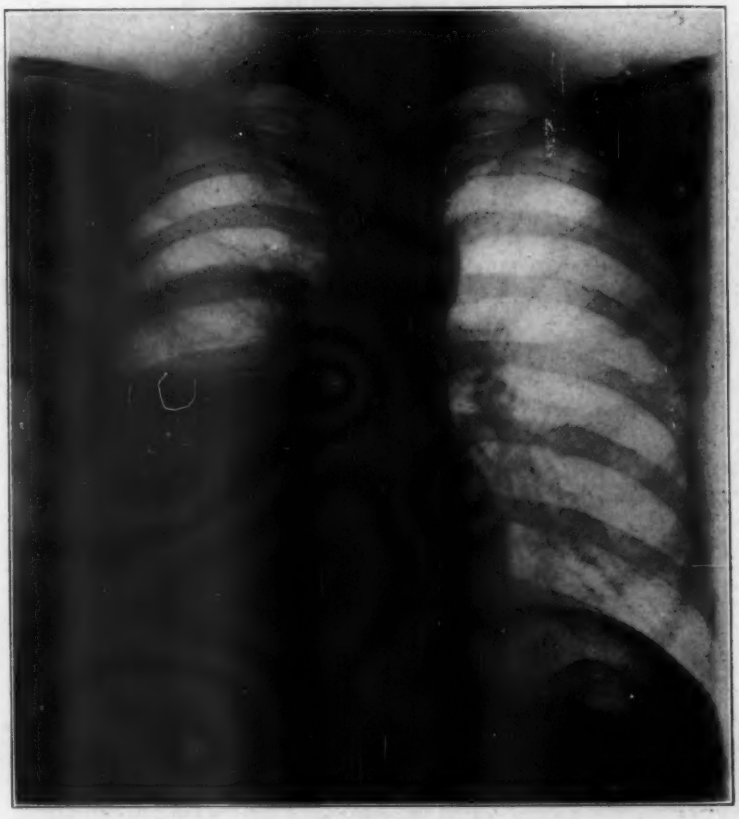

Fig. 11 (Case 3).—Condition April 11, 1920: sharply circumscribed upper border and lung markings may be seen through the air in the pyopneumothorax.

July 20, 1920: Very little change from the previous examination was noted.

CASE 3.—*History.*—C. W., white, man, physician, aged 35, during the influenza epidemic had all the symptoms of a typical attack in February, 1920, but apparently recovered. He had a second attack two weeks later.

Clinical Course.—After three or four days, marked cyanosis, dyspnea and leukocytosis were noted. He complained of pain in the right lower chest. Examination disclosed no signs of consolidation. At no time were there signs of consolidation. After three days, there was dulness in the middle of the right back in the scapular region. This rapidly became more marked, and

spread down until the right back from the fourth rib to the diaphragm showed marked dulness to flatness with diminished to absent breath sounds and fremitus. There were no signs of displacement of the heart.

Dull tympany, increased fremitus and bronchovesicular breathing were detected at the top of dulness. On paracentesis, no fluid was obtained.

The temperature dropped about 2 degrees, the patient's general condition became better and it was believed that the pneumonia was beginning to resolve (Fig. 11).

All signs, however, remained as described; leukocytosis, temperature varying between normal and 102 F., persistent pain in the chest and dyspnea, and limitation of motion of right chest. Paracentesis revealed thick pus. The signs remained practically the same from then until an operation was performed on April 15, 1920. There was never any displacement of the organs. Following the operation the temperature became normal and has remained so since.

The patient has now regained his normal weight and is doing heavy outdoor work without dyspnea or discomfort (Fig. 12).

Roentgen-Ray Findings.—The pathologic changes were confined to the lower part of the right chest. A uniform shadow extended from the spinal shadows of the chest wall, sharply limited above in a horizontal line and impinging laterally on another line evidently representing thickened pleura. The degree of density was not very great, in fact some lung texture could still be made out through the most dense part of this shadow. The pleural angle was obliterated and the diaphragm was elevated. Above this horizontal fluid line was a smaller area circumscribed by an indefinite curvilinear line which followed the posterior chest wall. It was continuous with the shadow in the axilla, and we believe the whole represented a circumscribed cavity containing both pus and air. The left lung showed smaller areas of exaggerated reticulum both centrally and peripherally, evidently a residue of previous pneumonic involvement. There was beginning retraction of the interspaces on the right side, below. The displacement of the heart, which was not marked, was toward rather than away from the side presenting the most marked pathologic change.

March 16, 1920: There was no change.

March 23, 1920: Very slight diminution in the amount of fluid was noted; otherwise the roentgenogram was identical with the first one.

April 12, 1920: Density of the lower part was increased and the line limiting the air bubble above was denser. There was distinct thickening of the pleura laterally.

May 14, 1920: The roentgenogram showed the drainage tube in place. There was comparatively little pleural thickening though there was a definite double pleural line extending from the base to the apex. The opposite side showed a fine pleural line over the apex. The entire cavity, which had a rather thick wall, had shrunk until it was about twice the size of a hickory nut. The limiting membrane was only slightly more vascular than the rest of the lung. It was not completely consolidated and was not sharply delimited.

July 26, 1920: The right diaphragmatic angle was somewhat obscured and hazy. The bronchovascular tree in this region was of the finely reticulated type and that in the lower half of the lung was distinctly more marked. With the exception of the slight fibrosis and pleural thickening at the right base, the lung had returned to normal. The residual changes would easily be overlooked on casual examination.

Case 4 (No. 241).—E. P. entered the hospital January 26, complaining of weakness and general malaise. His symptoms and signs were those associated with typical influenza.

Clinical Course.—Shortly after admission, he had a chill and the temperature rose to 102 F. On the following day there were signs of consolidation throughout the entire lower left lobe with a patchy involvement of the middle lung field. On the 31st, he complained of pain in the left side on breathing;

Fig. 12 (Case 3).—Condition July 26, 1920: except for the haziness in the lower part of the right chest, no residual changes are demonstrable. Patient has regained normal weight and is able to do heavy outdoor work.

a friction rub was heard and the side was strapped. February 1, he was extremely cyanotic; the respirations were rapid, and the pulse was of poor quality, varying in rate between 130 and 150. The white count at this time was 10,000. From the 1st until the 8th, the temperature fluctuated daily between 99.5 and 103 F. February 11, 2,300 c.c. of thin greenish pus was removed from the left chest, and from this fluid a hemolytic streptococcus was isolated. On the 17th, and again on the 26th, the chest was tapped and similar fluid obtained. On the 26th, air was demonstrated on aspiration.

Until February 26th, the daily maximum afternoon temperature was about 102 F. Following the tapping, the maximum temperature was 99.5 until March 5, when it again rose to 103. On the 7th, 1,000 c.c. of purulent fluid was removed from the left chest. The fluid on this occasion was considerably thicker than that obtained on previous aspirations. On March 9, he was transferred to the surgical department and on March 12, under local anesthesia, a rib resection was performed and two drainage tubes were inserted. Following this the patient made an uneventful recovery.

Roentgen-Ray Findings.—Feb. 25, 1920: The lower portion of the left thorax was filled with a uniformly dense shadow with an approximately horizontal upper level above which was a space free from any detail which evidently contained air. This air space was limited above by a curvilinear line reaching from the third rib in the axilla to the middle of the hilus, and

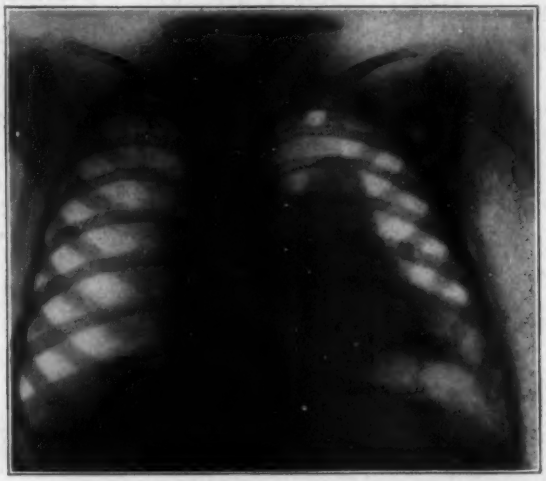

Fig. 13 (Case 5).—Condition March 1, 1920: unusual picture of the collapse of a consolidated lung. Without consolidation this lung might not be seen beyond the shadow of the sternum and spine.

below by a broadly curved line evidently representing some portion of the heart. The upper lobe on the left showed a great thickening along its base. This was evidently due to interlobar thickening The texture of this lung was exaggerated. The reticulum was marked and a few localized opacities were noted peripherally. The right side of the thorax showed some indefinite localized increase in the bronchovascular tree suggestive of a past influenza. The cardiac shadow was much displaced. The apex seemed to be on the right side, but a fine differentiation in this region was not possible.

March 7, 1920: The findings were almost like the preceding except that the amount of fluid was less than before, and the appearance of the left upper lung had lost all features suggestive of tuberculosis. The retraction of the interspaces was distinctly more marked and the thickness of the pleura had increased.

Case 5 (No. 5103).—*History.*—R. R., aged 2, girl, entered the hospital February 5, as an emergency case of influenza.

Examination.—This revealed signs of consolidation at the base of the right lung.

Clinical Course.—The patient improved until February 25, when signs of fluid on the left were discovered. At that time, the right chest was practically free from physical signs. Thoracentesis yielded a seropurulent fluid. The patient was again aspirated on the 29th. The patient struggled during this procedure and only about 1 c.c. of fluid was obtained. After this a small amount of subcutaneous emphysema developed (Fig. 13). On March 4, signs of air and fluid were obtained on the left. The heart was displaced to the right. The patient died during an attempt to aspirate the chest.

Necropsy.—This revealed a left pyopneumothorax with complete collapse of the lung which was covered by an exudate one eighth inch thick.

Roentgen-Ray Findings.—Feb. 26, 1920, abnormal opacity in the left lower lobe mostly behind and reaching to the seventh rib was noted. This opacity was diffuse, showing imperfectly the exaggerated reticulum with the density near the center completely obscuring the costophrenic angle. It did not rise characteristically in the axilla. There was no retraction of the interspaces or displacement of the heart or mediastinum. There was a distinct shadow corresponding in shape and position to a thymus of considerable size.

March 2, 1920: An unusual picture of an intensely opaque collapsed lung lying free in an air-containing pleural cavity was presented. The outlines of this lung were those of the expanded lung and there was evidently consolidation of both lobes on the left side. In addition there was a pneumothorax without demonstrable fluid in this position (roentgenograms were taken with the patient in the prone position). There was some displacement of the mediastinal contents but this was not very marked. The thymus shadow at this time was not very conspicuous. The border of the heart was entirely lost in the shadow of the lung.

COMMENT

These cases present almost all the conceivable findings in such a process. The first two cases are of interest because of the long duration of the empyema. Case 1 shows the strong tendency on the part of these patients to recover spontaneously. The treatment consisted only of rest in bed and relatively few aspirations. Cases 2 and 3 show also the tendency toward spontaneous recovery but in these cases improvement became stationary under expectant treatment. Both of these patients recovered, and the physical roentgenographic findings revealed rapid improvement after a simple thoracotomy and a tubular drainage. In Case 4 the patient recovered clinically after rib resection and drainage. Case 5 shows the importance of position in making the diagnosis of air and fluid by roentgen-ray examination and is perhaps added evidence in support of the proposition that in this form of bronchopneumonia it is not necessary to consider the empyema (unless it becomes so massive as to cause mechanical difficulty) until the acute process has subsided.

In two cases (1 and 3) the condition of the chest at the present time is practically indistinguishable from that of the normal. It is

not possible to say how much deformity will eventually remain in Case 2 but judging from the others a great deal of the pleural thickening should disappear. Unfortunately, we have no recent roentgenogram of Case 4 but clinically the deformity is not great.

It would, therefore, appear that expectant treatment with aspiration when indicated was justified in these cases until improvement was no longer clinically demonstrable. At this time, the indication is for the simplest surgical procedure that will supply adequate drainage.

We present these cases and roentgenograms to demonstrate how completely the normal chest markings may be restored even after the presence of extensive adhesions, marked pleural thickening and pulmonary deformity. In treating cases of this type, it should be remembered that during the acute stage the effusion should not be removed, unless there are definite indications. We believe that the procedures which are subsequently employed should be as conservative as possible. The indications for the employment of these should be determined by the clinical course and by frequent roentgen-ray examinations.

THE RÔLE OF THE CORTICAL LYMPHATICS IN THE EXTENSION OF TUBERCULOSIS WITHIN THE KIDNEY

E. GRANVILLE CRABTREE, M.D.

BOSTON

Clinical experience has established the futility of partial nephrectomy as a surgical measure in cases in which the lesion of the kidney is tuberculous. The purpose of this communication is to contribute further pathologic evidence as to the paths of extension of tuberculosis within the kidney, in justification of operations now employed, rather than to discuss what is already established as a sound surgical procedure.

The observations here recorded were made on a group of twenty-one kidneys, obtained at operation, which showed macroscopically a single small lesion at some point in the renal substance with an attendant tuberculous pyelitis. In some instances, the cortex was almost, if not entirely, free from gross lesions, yet in most instances there were a few small cortical tubercles to be seen overlying the gross lesion of the interior of the kidney and connected with it by lines of tuberculous infiltration. There was in each instance a considerable portion of the kidney cortex which seemed grossly to be normal. To these apparently uninvolved portions of the cortex my study was directed with the purpose of demonstrating new lesions, if any were present, which might be concerned in the subsequent destruction of the kidney. These early cases are not common. The twenty-one kidneys studied have been collected from the surgical services at the Massachusetts General Hospital and from private practice.

The studies of Ekehorn [1] have thrown much light on the manner in which a tuberculous focus in the kidney spreads in the pelvis of that kidney. It is generally recognized that a primary tuberculous focus in the kidney is almost invariably situated in the region of the medullary portion of the kidney cortex at the base of the pyramid in the region of the loops of Henle. That these may exist for a time without symptoms seems to be a clinical fact. Eventually the process extends in all directions and breaks by the shortest path spontaneously into the pelvis which is nearer than the cortical surface. After the tuberculous process has ruptured into the pelvis, a tuberculous pyelitis with its attendant symptoms soon develops. Ekehorn

1. Ekehorn: Die Ausbreitungsweise der Nierentuberkulose in der tuberkulösen Niere, Folia Urolog. **2**:412, 1908.

has demonstrated by serial sections of all the pyramids of such a kidney, that all the pyramids rapidly become infected. Tubercle bacilli are found in greatest numbers in the recesses of calices where urinary drainage is probably less complete, and the pyramids soon become involved in these regions. From these lesions, he believes ascending infection takes place within the kidney along the lumens of partially obstructed tubules and by way of the intertubular lymphatics and perhaps by the blood vessels.

The presence of tuberculous pyelitis is, in itself, sufficient evidence to justify the complete removal of a tuberculous kidney no matter

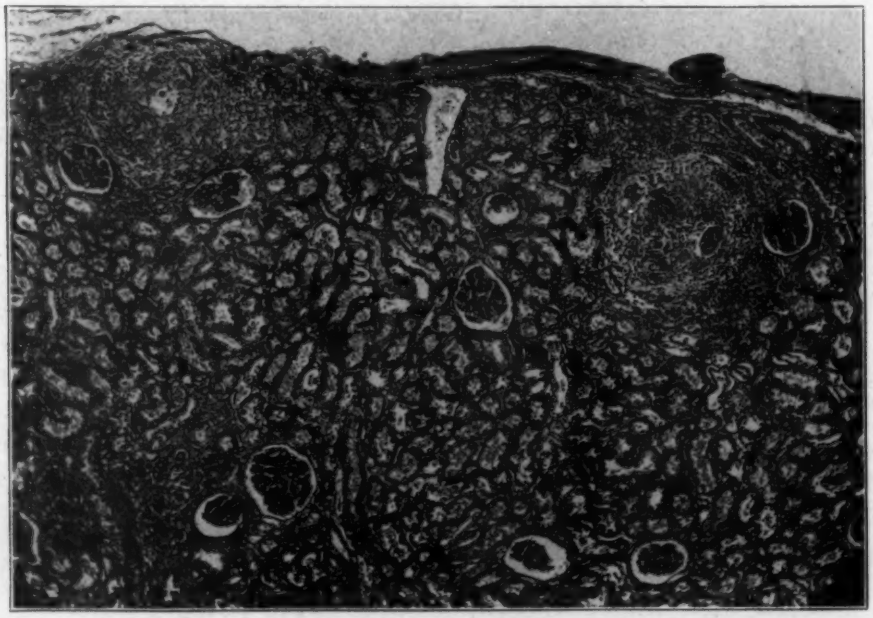

Fig. 1.—Microscopic section, × 50, from the apparently normal portions of a kidney in the opposite pole of which there is a small active tuberculous focus. Note that the tubercle apparently takes origin from the interstitial tissue and not from glomeruli. Aside from the row of cortical tubercles, two of which are shown here, the tissue of this pole of the kidneys shows no tuberculous changes. There is tuberculosis of the pelvis throughout all its parts. (Photomicrograph by L. S. Brown.)

how small the amount of renal tissue destruction which may have taken place. There is, however, a small group of cases in which there is a double kidney with separate pelves, only one of which is infected; another group in which there are fused kidneys, and more rarely a group in which there are normally developed kidneys in which a small chronic lesion of one pole has resulted in the formation of a sclero-

caseous partition which separates that portion of the pelvis from the remainder. In these cases the temptation to perform a partial nephrectomy is great. I have one double kidney in my series of twenty-one, only one pelvis of which shows a tuberculous pyelitis, yet the outlying cortical tissue of the portion connected with an uninvolved pelvis shows microscopic tubercles.

I have repeated the work of Ekehorn and believe that it represents the facts. Yet in many instances, I find in apparently normal portions of the cortex, remote from the region of the primary focus, numerous

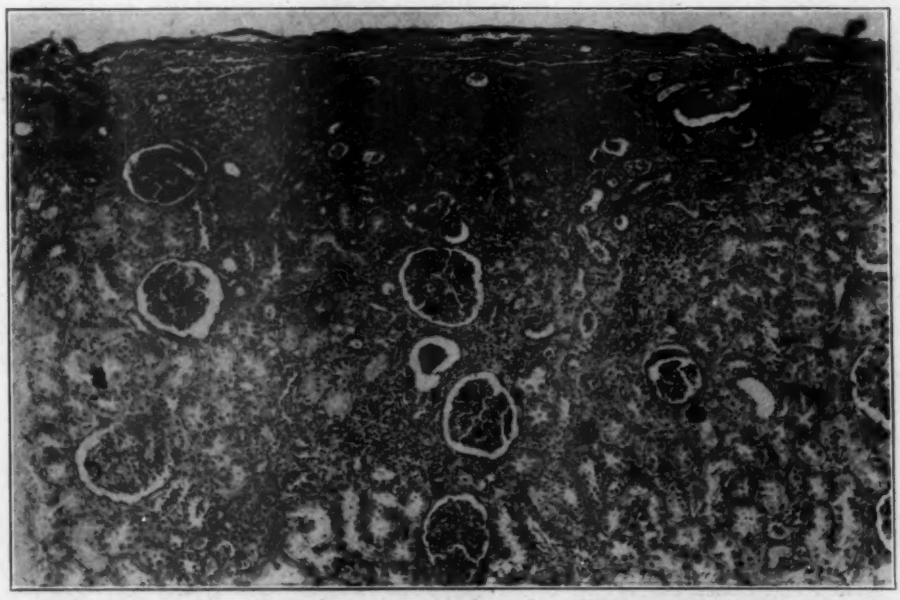

Fig. 2.—A small area of diffuse tuberculous infiltration, × 70, just beneath the capsule in a portion of a kidney macroscopically normal. There is a small focus of active tuberculosis in the opposite pole from where this section was taken. Note that the glomeruli are not involved. Tubercle bacilli were found in the region of the giant cells near the central portion of the area. (Photomicrograph by L. S. Brown.)

microscopic tubercles just beneath the true capsule which seem not to be connected with the pelvis by the usual fine lines of round cell and endothelial cell infiltration which is usually found where there is extension to the cortex from an underlying caseating cavity. Also in the one double kidney there was no demonstrable process in the pelvis or medullary portions of the other half, yet the cortex showed numerous small tubercles beneath the capsule. I believe that these lesions are due to lateral extension by way of the cortical lymphatics.

Kumita,[2] Sakata[3] and Sugimura[4] have demonstrated in the fatty capsule of the kidney two intercommunicating plexus of lymph channels which in turn connect with a lymph plexus just beneath the true capsule. Lymph channels from the parenchyma drain into the lymph plexus just beneath the true capsule and from that may communicate with those of the fatty capsule. Undoubtedly the intertubular lymph spaces play an important part in spreading infection between pelvis and cortex. With these spaces invaded, infection of the greater lymph spaces of the cortex is easily accomplished. By this means tubercle bacilli may be distributed along the cortex to portions of the kidney remote from the primary lesion; but since the lymph channels of the separate portions of a double kidney and of fused kidneys intercommunicate, cortical invasion of the second kidney may also be expected.

I am unable to offer an opinion as to how great a part these microscopic tubercles play in accomplishing the destruction of the kidney. The small cortical tubercle, indeed, is often found sclerosed. Yet I am not prepared to deny that secondary tubercles may not develop deeper in the kidney tissue from these cortical tubercles, and these secondary lesions be of considerable significance in accomplishing the destruction of the kidney. In one or two instances, I have found beginning tubercles containing bacilli a short distance down in the kidney substance directly beneath the cortical tubercle, situated in a medullary ray and connected with the tubercle by lines of tuberculous infiltration. The fact that the small cortical lesions actually do contain tubercle bacilli; that they may be found in portions of the kidney that are macroscopically normal, and that they may even extend across the line of division between the halves of a double kidney, seems to me to indicate an origin separate from the process in the pelvis.[5]

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2. Kumita: Ueber die Lymphgefässe der Nieren- und Nebennierenkapsel, Arch. f. Anat. u. Entwcklngsgesch, 49-58, 1909.

3. Sakata, K.: Ueber den Lymphapparat des Harnleiters, Arch. f. Anat. u. Entwcklngsgesch, 1, 1903.

4. Sugimura, S.: Ueber die Beteiligung der Ureteren an der akuten Blasenentzündigung usw, Virchow's Arch. f. Path. Anat. **206**:20, 1911.

5. The contents of this paper are embodied in the section on "The Pathology of Renal Tuberculosis" in Cabot's System of Urology.

ABDOMINAL INCISIONS*

E. P. QUAIN, M.D.

BISMARCK, N. D.

One of the important problems confronting the conscientious surgeon is that of gaining access to deep seated pathologic conditions in such a manner that the patient may suffer the least possible postoperative anatomic damage. It is true that our first duty is to save or prolong life, and to cure disease. But if in our efforts to accomplish these laudable things we leave the patient with a new pathologic condition of an equal or even more disabling character than the disease itself, then the surgical treatment was ill-advised or ill-managed, and the cause of surgery will suffer because of the fact that the patient continues to suffer.

A number of lesions amenable to surgical treatment are amenable only through the infliction of serious and permanent anatomic damage. This is often the case in the surgery of cancer and in various infections which threaten the patient's life or limb. In such instances, it would be a serious mistake not to act bravely even when mutilation is necessary. But the great majority of the surgical operations performed today are more or less of an elective type, and of such a nature that knowledge of anatomy and good technic should reduce to a minimum all unpleasant postoperative symptoms. In this class of surgical lesions we must in a certain sense reverse the rule we follow in radical cancer surgery, and we should say to ourselves, first, "We shall do no harm," and second, "We shall cure the disease."

The incision through the anterior abdominal wall in elective laparotomies demands considerable forethought with certain definite aims if we are to live up to our first dictum. We must have in mind these anatomic facts: The main body and strength of the abdominal wall is made up of three lateral, flat and superimposed muscles, the external and internal oblique and the transversalis. These muscles take origin from the ribs, lumbar and iliac bones, and end in one broad tendon or aponeurosis which unites at the midline with its fellow of the opposite side (Figs. 1 and 2). This tendon is split vertically on each side of the midline to form a canal within which lies the practically sheathless rectus muscle. At the outer margin of this canal is the semilunar line

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* Read before the Montana State Medical Society, Helena, Mont., July 14, 1920.

or conjoined tendon of the lateral muscles. It is important to recognize this landmark in all lateral incisions into the abdomen. The function of the three lateral muscles is to pull outward and backward from the midline in front and thus to produce a narrowing of the abdominal cavity and an increase in intra-abdominal pressure. Contraction of the rectus muscle in front tends to shorten the abdominal cavity by approximating the ensiform cartilage to the pubes. It has but loose fibrous connections with the aponeurotic tube within which

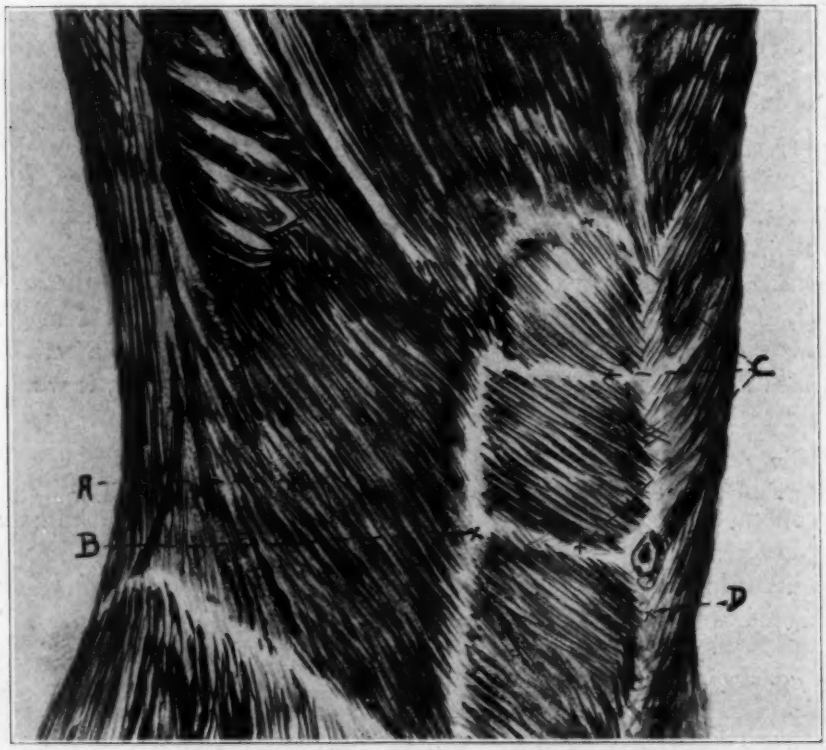

Fig. 1.—Outer layer of muscles and aponeuroses of anterior abdominal wall: *A*, external oblique; *B*, linea semilunaris; *C*, lineae transversae, and *D*, linea alba.

it lies and can therefore retract an appreciable distance when cut across, especially in its lower half. Under the muscles lies the transversalis fascia. The fibers of this structure also run transversely and parallel to the transversalis muscle and carry with them in close association the peritoneal blood and nerve supply. The peritoneum is more closely attached to the transversalis fascia above the umbilicus than below.

The blood supply is derived from the intercostal arteries outside the semilunar lines and from the deep epigastrics between the two semilunar lines (Fig. 3).

The nerve supply to the abdominal wall so far as known is derived entirely from the intercostal nerves, usually from the sixth to the twelfth. These nerves control the functions of the muscles and carry sensations from the skin and peritoneum. They have the same trophic relationship to the structures of the abdominal wall that the brachial plexus has to those of the hand. The intercostal nerves enter the abdominal wall at the costal border, between the internal oblique and transversalis muscles. From this region is given off a very fine network of nerve filaments which pass mesially to supply the peritoneum. The coarser and visible nerve fibers, running more or less parallel to the muscle fibers and aponeuroses, end in these structures and in the overlying areolar tissue and skin. The motor nerves leading to the rectus muscle pass underneath the outer margin of this muscle and

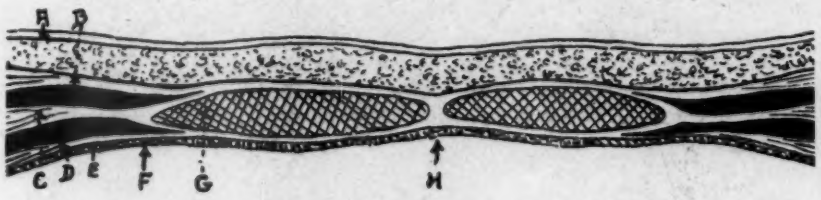

Fig. 2.—Transverse section of abdominal wall below umbilicus: *A*, skin; *B*, external oblique; *C*, internal oblique; *D*, transversalis muscle; *E*, transversalis fascia and peritoneum; *F*, opposite semilunar line; *G*, rectus muscle, and *H*, linea alba.

enter it along its central line near the deep epigastric artery (Fig. 4). From this line, the nerve filaments are distributed both outward and inward to supply the rectus muscle.

Keeping these anatomic relations in mind, it is evident that whenever an abdominal incision is made vertically, that is, parallel to the long axis of the body, the structures which give the chief support and tone to the abdominal wall are being cut across. A vertical incision in the median line, while it temporarily loosens the insertions of the lateral tendons, can fairly easily be restored almost to normal. But if the vertical incision be made to the side of the midline, as is the case in the transrectal or pararectal incisions, then an anatomic injury has been inflicted which in all likelihood cannot be restored to normal. The fibers in the aponeuroses, that is, the tendons of the lateral muscle bundles, are severed, and while it is true that they may be resutured and made to heal, it is also true that very often they do not heal firmly

enough to withstand the intra-abdominal pressure. The cut fibers are pulled away from the wound by the lateral muscles, making proper peritoneal closure very difficult, as all those know who have seen the catgut tear out between the fibers while they were trying to close vertical incisions on patients just recovering from deep anesthesia. Pannett [1] found experimentally that any irritation of peritoneum or viscera produced an active spasm of the lateral muscles. This spasm may continue for some time after the operation, and the usual plain catgut closure is not enough to insure against a later separation of tissues.

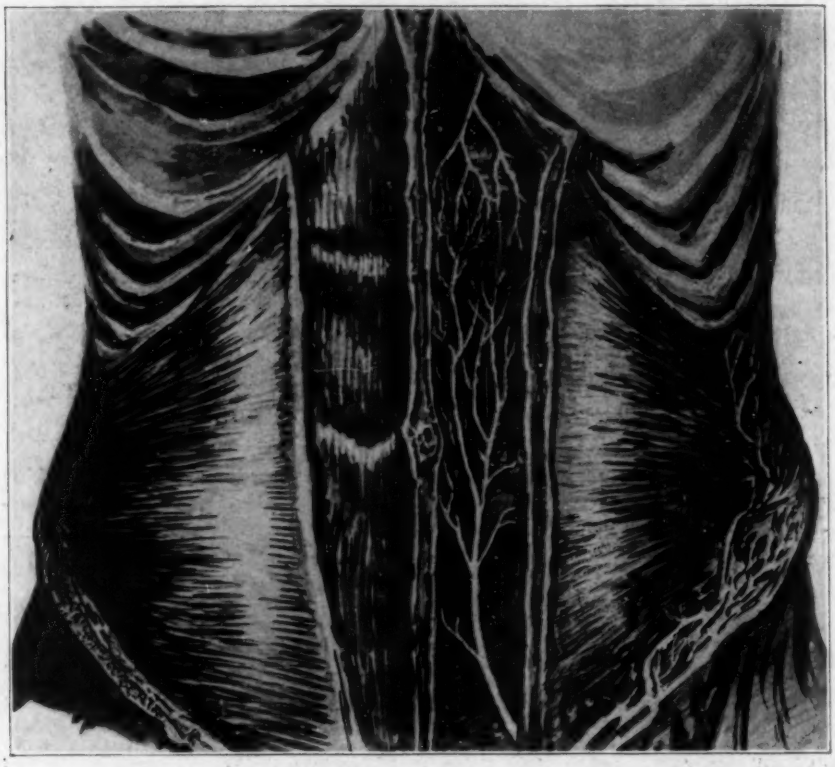

Fig. 3.—Left rectus removed, showing distribution and anastomosis of internal mammary and deep epigastric arteries.

When it becomes necessary to leave drainage because of infection in any vertical wound, there is a permanent gap between the fibers and postoperative hernia and other troubles are always invited. After drainage has been left in situ for a few days, the ends of the fibers become fixed in their retracted position by new tissue, and there is no physiologic force tending to re-approximate them—quite the contrary.

1. Pannett, C. A.: Surg., Gynec. & Obst. **30**:408 (April) 1920.

Another serious injury inflicted by the lateral longitudinal incision is the destruction of the intercostal nerve supply to the structures between the incision and the midline, or linea alba. This has been found in many cases to be of greater significance than the harm described in the aponeurosis. If two or more intercostal nerves are cut, the paralysis in their segment of the rectus muscle becomes permanent. The muscle atrophies, and becomes fibrous. Unless strong aponeuroses are present which heal very firmly after the operation, and unless the patient exercises special care in the use of the abdominal muscles after operation, there is likely to be more or less bulging at

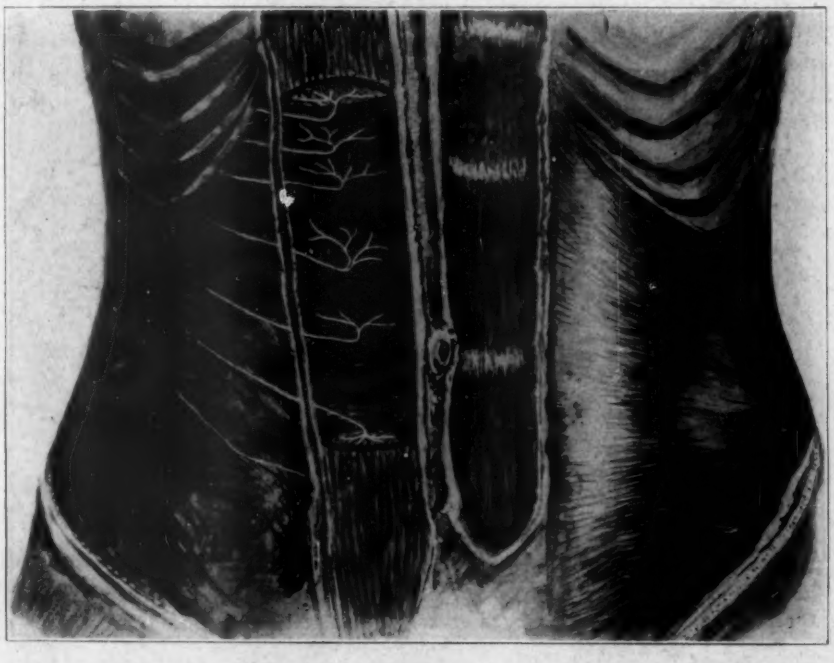

Fig. 4.—Segment of right rectus removed, showing intercostal nerves entering rectus muscle along its posterior central line.

this part of the abdomen a few weeks, or months, later. It is possible to incise the aponeurosis behind the rectus and preserve the visible nerve fibers running across the line of incision. This may save the rectus muscle, but it does not save the peritoneum for its nerve supply originates from the intercostal nerves at the costal margin; and the filaments follow the transversalis fascia as a delicate network, imperceptible to the naked eye. Severing the peritoneum thus from its nerves removes the trophic influence required for the maintenance of normal physiology. If the enervated section of peritoneum has been bruised or mildly infected during the operation, it is less able to endo-

thelialize properly and to overcome infection than is the normal serosa. The result is that permanent adhesions of omentum or other organs are often found over part or all of the enervated area. At one time I [2] proved this clinical observation in a series of animal experiments. In these experiments it was found that a section of peritoneum deprived of its intercostal nerve supply is unable to overcome an amount of trauma or infection which a normally functioning peritoneum would take care of, and leave no permanent evidence of damage.

AUTHOR'S METHODS

In order that the incision itself might leave the least possible pathologic residue and at the same time be well placed, ample, and offer the most satisfactory line of drainage, for several years my associates and I have used the methods here described:

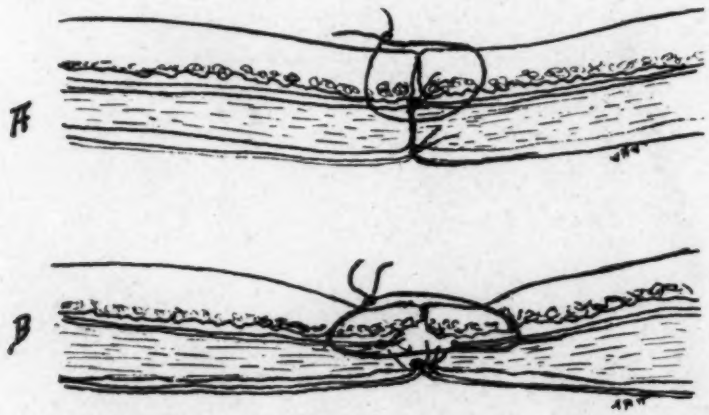

Fig. 5.—*A*, side-to-side silkworm gut fixation sutures; *B*, inefficiency of this suture to hold edges of aponeuroses together against lateral muscle strain.

The median incision is the routine in most pelvic operations. The Pfannenstiel or transverse incision is sometimes preferred when the round ligaments of the uterus require surgical intervention. It is of special advantage in the presence of a very fat or short abdomen. When it is known beforehand that infectious accumulations in the pelvis will require drainage after operation, it has been found wise to establish vaginal drainage through the culdesac before the abdominal incision is made. This will often obviate all soiling of the wound from a sudden gush upward when the abscess cavity bursts open.

In closing the median incision, special care must be given to the approximation of the deep aponeurosis at Douglas' fold. Several

2. Quain, E. P.: Am. J. Surg. **28**:133 (April) 1914.

hernias have been seen because this point had been overlooked. The two recti should be sutured together to reinforce the linea alba. The anterior aponeurosis will heal most surely if the margins are overlapped one-fourth or one-half inch. Edge-to-edge approximation is not enough. Softening of the catgut and straining of the muscles favor separation from the moment the operation is completed. The side-to-side silkworm gut sutures, usually depended on for fixation of the wound during the healing process, do not hold the wound firmly together unless they are tied so tightly that they cause injurious pressure on the tissues (Fig. 5). A more effective method, and a method

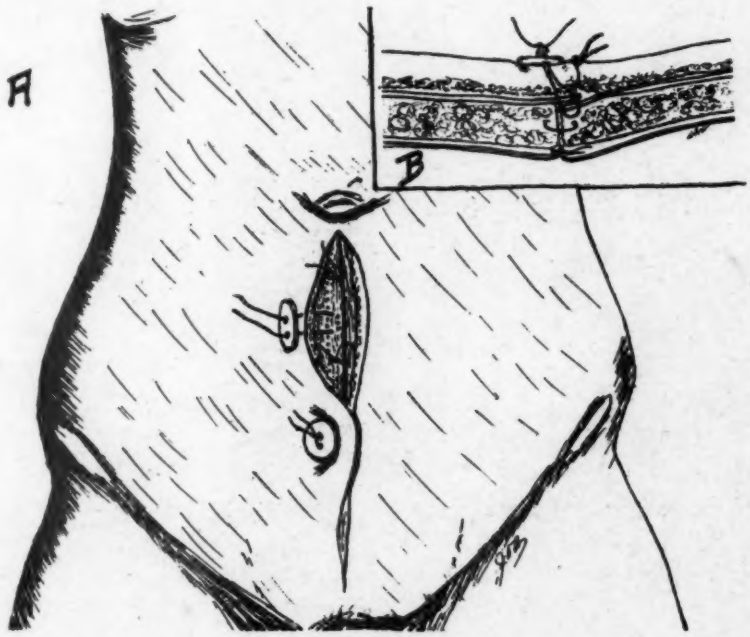

Fig. 6.—*A*, closure of median incision, showing overlapping of anterior aponeurosis and position of silkworm gut fixation sutures: *B*, cross-section of closed wound.

less constricting to tissues, is the introduction of a silkworm gut through the overlap of the aponeuroses in a short bite only, the two ends of the suture coming out closely together, well to the side of the wound, and being tied over a suitable button. The two aponeurotic surfaces coming in contact should be free from all intervening tissue, but for the purpose of insuring immediate blood supply to the margins of the aponeuroses it is advisable not to loosen the undersurface of the under flap from the rectus muscle, nor the upper surface of the upper flap from the fat and subcutaneous tissues (Fig. 6).

The appendix and the lower ureter are accessible in most cases through the so-called "split muscle" incision, placed somewhat below McBurney's point. The fibers of the external oblique should be split far enough both up and down to permit easy retraction of the slit in the two deeper muscles and in the transversalis fascia. The situation of the motor nerves for the inguinal region, just inside the anterior superior spine, must be remembered whenever the transversalis fascia is split far outward.

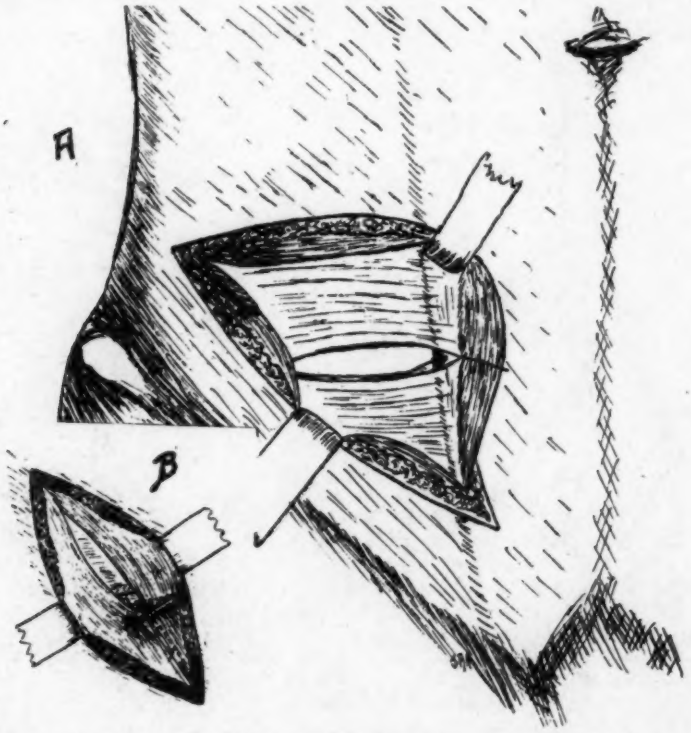

Fig. 7.—*A*, split muscle incision extended across semilunar line: *B*, closure of incision.

When pus is expected to escape through the wound, care should be exercised that the space between the external and the internal oblique be not opened farther than absolutely necessary. The infection will burrow as far as this space is opened, and it may go farther.

If the strictly "split muscle" incision is found to be too small, it can easily be enlarged by cutting across the semilunar line and separating the fibers of the aponeurosis over the rectus and retracting the rectus (Fig. 7). If more space is required, the rectus muscle will suffer no harm from being cut across and later resutured. If the original oblique incision was not too high and the wound is thus

enlarged mesially, the right half of the pelvis is easily accessible to the operator. Should the tip of an abnormally situated appendix be adherent high outside or behind the ascending colon and therefore inaccessible through the low opening described, it can nearly always be reached by extending the incision between the external oblique fibers outward and making a second splitting through the deeper muscles and the peritoneum (Fig. 8). To increase the accessibility in this manner by means of a second peritoneal opening is a much less destructive procedure than to yield to the temptation of enlarging the original wound by cutting across the lateral muscles and the transversalis fascia with the intercostal nerve fibers.

Fig. 8.—Secondary splitting of internal oblique and transversalis muscles at a higher level, to expose upper part of rectocolic space (usually made much higher than indicated here).

In the upper abdomen, the most important organs requiring surgical treatment are the biliary apparatus and the stomach. Our preference in stomach surgery and our routine in operations on the gallbladder and ducts, is the straight transverse incision, made about 2 inches above the level of the umbilicus (Fig. 9). The incision is carried down through the aponeurosis from a point near the linea alba and outward to, or beyond, the linea semilunaris. Only the outer fibers of the rectus muscle are cut at first. The posterior aponeurosis near the semilunar line is exposed and split. The transversalis muscle and fascia with the attached peritoneum is opened sufficiently to admit

one or two fingers for preliminary exploration. The incision is then continued to the costal margin, or across the linea alba and left rectus, as desired. Two or three spurting arteries may need ligation near the posterior surface of the rectus muscle. Another artery requiring ligation runs in the falciform ligament of the liver which is usually severed whenever the incision extends beyond the midline.

The exposure of the organs mentioned will be found very satisfactory. After a few experiences the surgeon becomes convinced of a better exposure, an easier closure, and what is still more to be

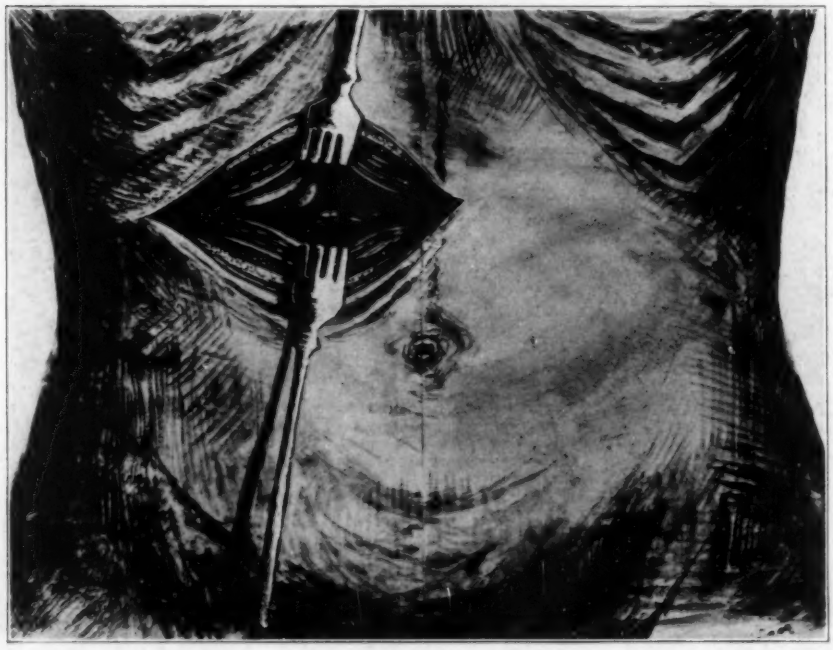

Fig. 9.—Transverse incision in epigastrium. Margin of liver and tip of gallbladder appearing in wound.

desired, he will learn that less harm has been done to the patient than by former methods.

Closure of the wound is accomplished by a running plain catgut suture of the posterior, and an interrupted, slightly overlapping, chromic catgut suture of the anterior aponeurosis. The cut ends of the recti may be approximated separately by a plain catgut suture if they seem to have retracted too far; but in most incisions they will be seen to come together very well from the interrupted suture of the external aponeurosis, the sutures being passed somewhat deeply into the muscle. An important point in the closure of this wound is the reconstruction

of the linea alba. This is best accomplished by a slight overlap, which overlap is reinforced by a silkworm suture in most of our operations (Figs. 10 and 11).

In a previous article [3] on "The Transverse Incision in the Upper Abdomen," my observations were based on slightly less than 300 operations by this method. Since that time our experience has been increased by more than 200 additional transverse incisions, about 400

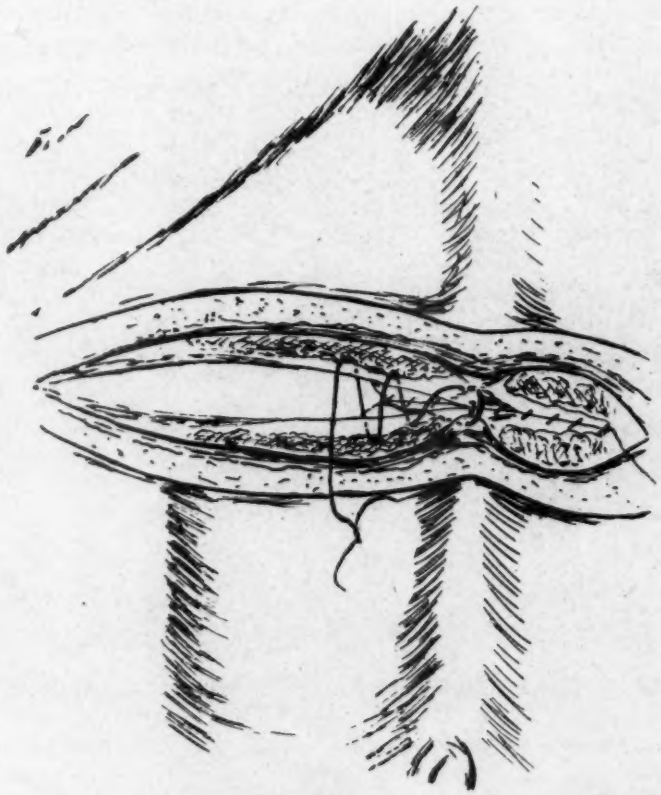

Fig. 10.—Closure of peritoneum beginning at left angle of incision, tied at linea alba, falciform ligament reconstructed and suture continued toward the right.

of the total number being made because of a pathologic condition of the biliary tract and 100, in round numbers, for gastric lesions. The favorable opinion expressed at that time both as to convenience of technic and after-results seems more than justified after this wider experience. The comparative freedom from pain in the wound, the cosmetic appearance of the scar, and the absence of hernia have been

3. Quain, E. P.: Journal-Lancet **37**:657 (Oct. 15) 1917.

almost constant among our postoperative observations. One patient in whom an unusually extensive drainage had been employed because of a ruptured empyema of the gallbladder returned after two months with a distinct hernial bulge over the outer part of the rectus muscle. There seemed to be a defect in the deeper structures nearly an inch in diameter. Operation for its closure was advised, but the patient was unable to spare the time because of urgent farm work and agreed to return two months later. He did so and at that time it was found that the bulging had become distinctly smaller. Operation was again postponed two months for the purpose of studying the unusual behavior of this ventral hernia. When he returned at

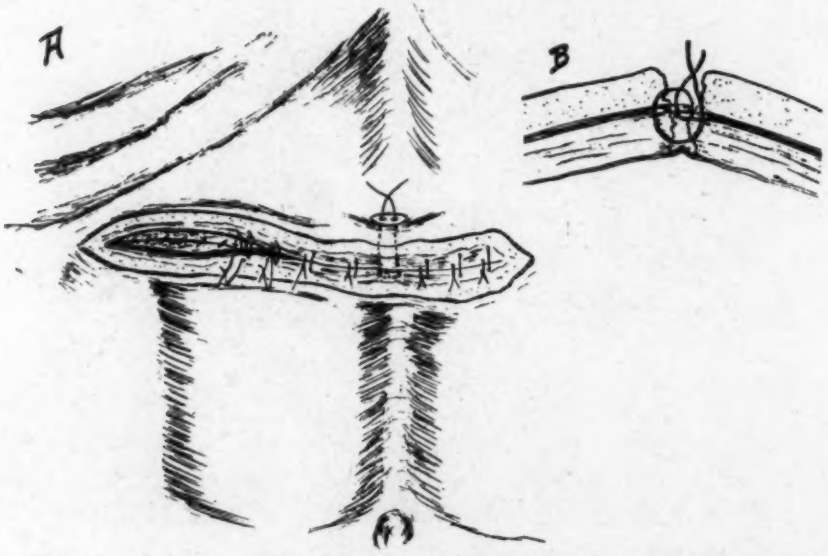

Fig. 11.—*A*, interrupted suture of rectus muscle and external aponeurosis, making the latter overlap. Silkworm gut suture through skin for fixation of linea alba. *B*, method of passing catgut through aponeurosis and rectus muscle.

the end of that time, there was no bulging at all. A spontaneous cure of an incisional hernia had taken place. One transverse wound burst open seven days after operation. This occurred in the case of a soldier on whom a cholecystectomy had been performed for empyema of the gallbladder in the presence of a severe bronchitis. The patient had a rather stormy recovery after operation because of the lung infection. The ward surgeon who had a standing instruction to remove all superficial stitches on the sixth day, because of his surgical inexperience, removed also the two silkworm gut fixation sutures on this day. The next day, in a severe fit of coughing, the wound suddenly gave way, and part of the omentum protruded. Immediate operation saved the patient's life.

From clinical experience, I have accumulated a large amount of evidence showing the harmful effects of lateral vertical incisions. During my service at a cantonment hospital, there were seventeen draft men operated on for hernia, pain, and adhesions in the scar after previous operations for appendicitis. In some of these, the appendix had been removed; in others, there had been only a simple drainage of an abscess. It was noted that in fifteen of the seventeen, either a straight right rectus incision had been made, or else an oblique incision with such an inclination downward along the semilunar line as to cut across the aponeurosis to some extent. Only two of the seventeen had had McBurney or split muscle incisions. In one of

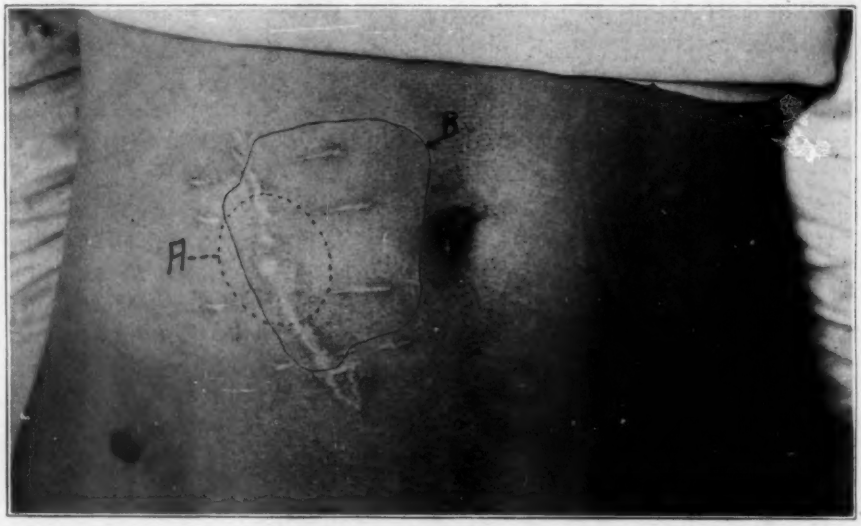

Fig. 12.—Patient with bulging and peritoneal adhesions after atypical right rectus incision (wound having healed primarily). *A*, outline of bulging due to defect in aponeurotic closure; *B*, outline of visceral adhesions to parietal peritoneum enervated by the incision.

these, there were three separate small hernial sacs, with adherent omental tags between the transversalis fascia and muscles. In the other was found a loop of ileum firmly attached to the peritoneum and transversalis fascia. Since both had been clean cases, it is evident that they were both due to gross technical errors in the closure of the wounds; defective peritoneal suture in one, and catching of the bowel with one or more sutures in the other. Of the fifteen vertical incisions, two had healed primarily, but the hernia had appeared from heavy lifting, one and two years, respectively, after operation. It should be remembered that the base hospital received all defectives from a training camp which contained constantly from 40,000 to

70,000 recruits. We cannot believe that the rectus incision had been employed eight times more frequently than the McBurney among the many recruits who had been operated on for appendicitis before being drafted. If we should assume, in the absence of statistics on the subject, that the two incisions had been employed with equal frequency in the camp population, these figures would still indicate that the McBurney incision is more than eight times more satisfactory than the vertical.

In the reconstruction hospital where I was on duty after the armistice, there were seven soldiers operated on for incisional hernia. Two of these had followed appendectomies through pararectal incisions, one of which had been drained, while the other had been closed and healed primarily. Two hernias had followed operations on the gallbladder through long transrectal incisions, and one of these two patients had already submitted to three unsuccessful operations for the hernia. The other three hernias had followed atypical abdominal incisions for gunshot wounds of the abdomen. All these cases gave especially beautiful demonstrations of the degenerating effect produced both on muscle and peritoneum by the destruction of the intercostal nerve supply. The segment of the rectus muscle lying within the zone of the incision was atrophied and lifeless, and the peritoneum could not be distinguished because of firm adhesions (Fig. 12).

A review of our operations in private practice for ventral or incisional hernia following laparotomies at the hands of other surgeons has furnished us with the same proof. Nearly half of all such hernias have resulted from the type of incision denounced in this article. In many of these the degeneration of muscle, fascia and peritoneum in the enervated segment of the abdomen has been easily demonstrable and sometimes irreparable.

REVIEW OF CLINICAL HISTORIES

It is a rule in our practice that all patients who have had drainage after operations for suppurative appendicitis on leaving the hospital are informed that they may expect a hernia at the place of drainage. They are further advised to return for examination and treatment as soon as any abnormal bulging appears at the site of incision. It is conceded that data based solely on the number of patients who have returned for such secondary observations may not lead to entirely accurate conclusions as to the results following any given incision. Yet our clientele is fairly constant, and it is believed that a review both of primary operations and secondary findings in a series of patients gives information of distinct value.

Our review of clinical histories covers 347 consecutive cases of acute suppurative appendicitis, with localized or progressive peritonitis.

Appendectomy was performed in nearly all, and drainage was established in every case. The tubes or cigaret drains ranged in number from one to ten. Patients who died after operation are not considered. In 287 of the 347, a split muscle, or McBurney incision, had been

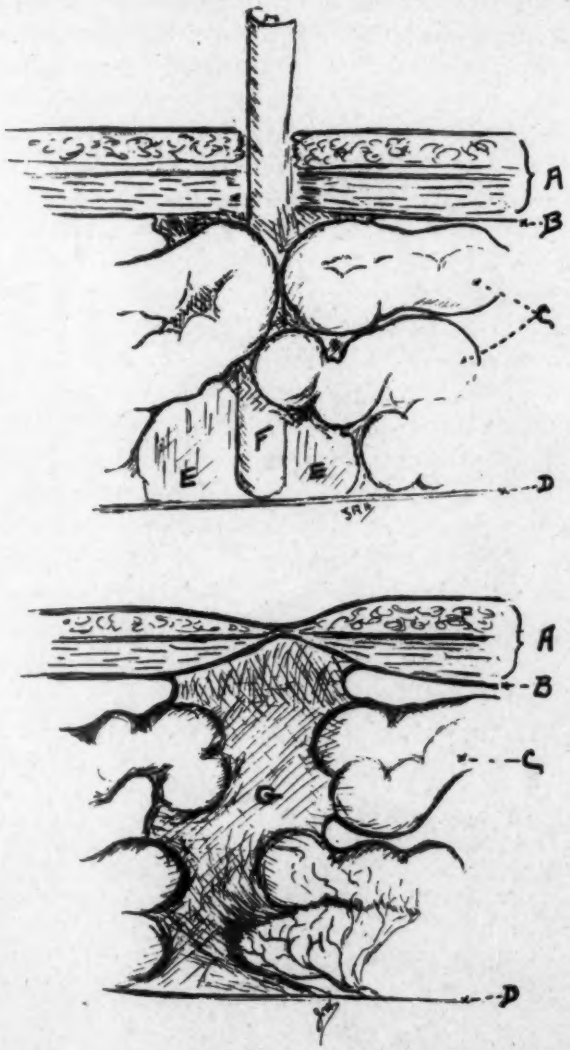

Fig. 13.—Result following drainage near center of abdomen: *A*, anterior abdominal wall; *B*, peritoneum; *C*, intestinal loops; *D*, posterior abdominal wall; *E*, abscess cavity; *F*, drainage material; *G*, postoperative adhesions, and *H*, mesentery.

used in the primary operation. This incision had been enlarged by cutting across the linea semilunaris in many instances. In a number of patients with extensive peritonitis, there had been a second split

muscle incision for drainage above the left groin. Twenty-six, or 9 per cent. of the 287, have returned for secondary operation for incisional hernia. In thirty-seven cases, the original split muscle incision had been found unsatisfactory at the primary operation, and it had therefore been enlarged by cutting across internal oblique and transversalis fibers, usually along the linea semilunaris, for from one-half to 2 inches. Nine of these, or nearly 25 per cent. of the number, have returned for operation to close the resultant ventral hernia. In nineteen cases, mostly children, a median or paramedian incision had

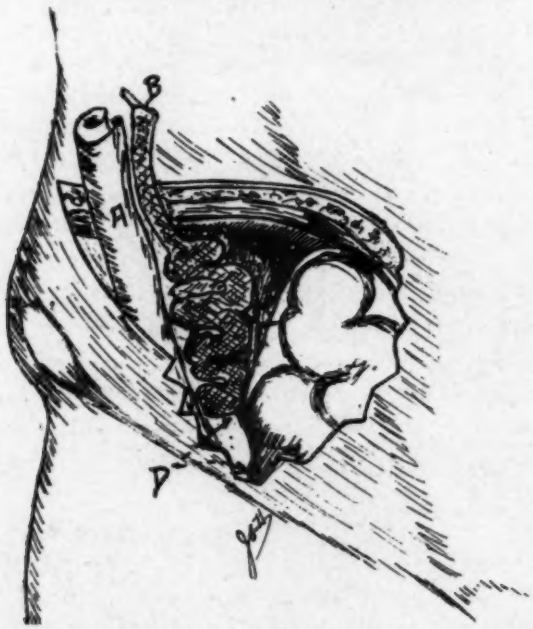

Fig. 14.—*A*, drainage near the groin and pushing of the peritoneum and transversalis fascia *C* by means of a gauze or rubber plug (*B*) help to shorten the intra-abdominal part of the drainage tract and thus to minimize the amount of postoperative adhesions *D*.

been made, and of these six, or nearly one third, have returned for hernia operation. In four patients, the gridiron opening had been made too low, and a second splitting of the internal oblique and transversalis muscles had been made near the crest of the ilium through an extension outward of the same incision for the purpose of reaching and draining high infection. None of these have returned.

Another observation made in the secondary operations after appendiceal drainage has been the comparative ease of repair in those cases in which neither muscle nor nerve continuity had been damaged by destructive incision. The peritoneal adhesions are less extensive and

may be absent. The muscle fibers above and below the incision have, through functional activity, liberated themselves to an appreciable extent from the postoperative interfascicular infiltration. The muscle planes are readily found and restored. After primary vertical incision, the ends of the severed muscle fibers have retracted en masse and are often found embedded in a mat of cicatricial tissue, requiring much surgical skill and judgment in their successful dissection and resuturing. The intra-abdominal adhesions also are as a rule much more extensive and are less likely to be limited to the scar area, than they are when the previous incision has been more anatomically made.

The introduction of sutures to close partly an incision after the whole wound has become contaminated in the operation should be disapproved. Such suturing favors direct dissemination of infection into previously sterile tissues and invites further extension of infection and necrosis by restricting free circulation of blood and lymph. Experience has shown that the sutures often slough away, and the sloughing may involve fascial structures which the abdominal wall can ill afford to lose. If the incision, or incisions, be made parallel to the functional forces transmitted by the muscle bundles above and below the wound, the edges will tend to approach one another after the drainage tubes are removed. Later this secondary and imperfect union may of course be disrupted by the increasing intra-abdominal pressure due to early resumption of occupational activities, yet the experiences cited above go to show the comparative infrequency of this occurrence.

Operations for intra-abdominal infections usually include, and may be limited to, the establishment of drainage. Physical law and surgical experience both agree that liquids escape from a cavity most readily if it is opened at the bottom. This was one of the reasons which led to the establishment of low pelvic drainage and the sitting posture especially for infections in the lower half of the abdomen. The drainage may be established in the midline above the pubes, or still better, through an incision above one or both groins through a separation of the lateral muscle fibers. The establishment of any form of drainage is always followed by visceral adhesions at the place of drainage for a longer or shorter period. If the drainage is made some distance away from the lateral abdominal walls, these adhesions will *surround* the place of drainage and later form a fibrous band uniting the anterior to the posterior abdominal wall (Fig. 13). The small intestines are frequently hampered in their free movements around this abnormal band of adhesions, and chronic pain or acute obstruction is not an uncommon result. By placing the drainage near the flank, groin or bladder, so that one side of the drainage tract

is made up of normal and permanent abdominal wall, the amount of adhesions possible is reduced by nearly one-half. The margin of the peritoneum can be loosened slightly from the overlying muscles and pushed downward with a small pack so as to shorten materially the intra-abdominal part of the drainage tract and thus lessen further the amount of adhesions (Fig. 14). From this technic there will be no postoperative band spanning the otherwise free abdominal cavity, and the patient will have less disability and less discomfort to remind him of his experience at the hands of the surgeon.

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